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WAR-NEURASTHENIA, ACUTE AND CHRONIC.

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Author's Note.

The material used in this paper was collected during 1917 and the paper itself was written at the end of that year, when there appeared to be an indefinite prospect of hostilities before us. I have thought it best to let it stand as written. In most writings dealing with this subject great emphasis is laid on the psychical side of it, sometimes I think to the neglect of the physical side. I have here attempted to deal with the latter in monographic form, and to describe war-neurasthenia as a a physical disorder, with symptoms affecting most of the chief systems. I was unable at the time of writing to obtain access either to current or standard medical literature, hence the absence of references. The tentative observations on the importance of the state of the ductless glands in the condition, which now appears to be generally accepted, would have been modified in any fresh consideration of the problem; at that time little was heard about them.

Introduction.

There are many terms in common use in medicine which convey popular ideas with sufficient clearness, but which are not scientifically exact. Such words are "rheumatism" and "influenza," meaning respectively, pains, referred to the body or limbs; and headache, malaise and the accessory symptoms which accompany a mild general toxæmia, without more localizing signs than catarrh. One of the latest additions to the list of popular medical terms is "shell-shock," which includes all nervous disease induced by military service without obvious organic lesion. Among such are to be found all the usual manifestations of

functional nervous disease, and on this account I have preferred the term "war-neurasthenia" as the title for this paper. The following pages are the result of a clinical study of these disorders, carried out under the following conditions.

In January, 1917, a centre was opened for the treatment of the cases of "shell-shock" from the 1st and 2nd Armies in France at No. 4 Stationary Hospital, first under the command of Colonel Bate, C.M.G., A.M.S., and afterwards of Lieutenant-Colonel Inkson, V.C., D.S.O., R.A.M.C.; general supervision was kept by Lieutenant-Colonel Gordon Holmes, C.M.G., Consulting Neurologist to the B.E.F.

The wards, and afterwards the Medical Division, allotted to the patients were in my charge, and for several months Captain A. E. Evans, R.A.M.C., assisted me. Other officers who served in the department for considerable periods were Captain J. Allan, R.A.M.C., Lieutenant W. L. Stewart, R.A.M.C., and 1st Lieutenant W. B. Terhune, U.S.M.O.R.C.

The department was open for nine months, during which period 4,700 patients passed through it; their records form the material on which this account is based. In the early part of the year patients were not numerous, and the majority of them were examined by myself, others by Captain Evans, working on a scheme drawn up by me, and in the same tent. Under this arrangement the first 1,300 patients were all examined in my presence, and subsequently treated and discharged by me; this series of cases has been used for statistical purposes and general close examination, though the results are interpreted in the light of the larger experience which was gained later.

The general object for which this and other centres were established was the return to duty of all possible patients, and the broad rule commonly observed, subject to the exigencies of the Service, was to send back all patients who became fit for duty within a month, and any who at the end of that time appeared to be still unfit were transferred to a base hospital.

The following account is a plain clinical study of the subject made by a general physician, with sufficient experience in disease of the nervous system to be able to make accurate diagnosis between organic and functional conditions. To ensure uniformity all patients were examined on a definite plan, in a special tent set apart for the purpose in which not more than two medical officers worked. A history of each case was taken, in which notes were made on the following points:—

(1) Name, number, unit, civil calling, age, total service, active service.

(2) *Personal "nerve,"* as commonly estimated, capacity to work on heights, manage horses, and the like; games and sports followed; history of "nervous breakdowns." "Nerve" is a slang word in this sense, but it expresses the meaning required.

(3) *Previous strain*, period and character of active service; previous wounds or shock, and their effects.

(4) *The shock*, if any; history of being blown up or buried, or of gradually oncoming failure.

(5) *Symptoms:* Inquiry was made as to pain, and as to obtrusive subjective symptoms, as to appetite, digestion, bowels; micturition; respiration; sleep.

(6) *Physical signs:* The patient was stripped completely naked and notes made of his general development, nutrition and strength. A routine examination was made of his thorax and abdomen; he was put through movements to test the integrity of the motor and co-ordinating systems, and the chief reflexes were examined. The sensory system was as a rule ignored; no search for anæsthetic or hyperæsthetic areas was made because there is no reason to suppose that an anæsthesia of which a patient is unaware will affect his efficiency as a soldier, whereas one to which his attention is drawn may easily become a source of morbid interest.

Since the symptoms complained of in these cases are chiefly nervous a scheme has been drawn up for their classification on a nervous basis. The central nervous system is considered as separated into somatic and splanchnic divisions. The somatic division is separated into its usual components of sensory and motor systems, due note being made of the parts of each system on which the function of co-ordination depends. Every part of these systems has its proper physiological function, and when this function is disturbed symptoms arise. The functions may be disturbed in two ways, either by way of excess or by way of defect; thus on the sensory side pain is considered as sensation in excess, anæsthesia as sensation in defect; on the motor side contraction in excess is spasm, either tonic or clonic; contraction in defect is paresis; on these lines the list of symptoms has been constructed. A similar but less elaborate scheme for the splanchnic division has been employed in which note is taken of subjective and objective symptoms in the circulatory, respiratory, alimentary, and excretory systems.

The patients had all passed through a field ambulance and a casualty clearing station before admission; consequently all cases of shock were seen at least one, generally two, and often more days after the onset of symptoms, so that no experience was gained of the immediate conditions produced by shock; but the cases were as a rule of recent onset.

In view of the practical necessity for evacuation at the end of a month there was a similar lack of experience with regard to the late results in severe cases. Under a General Routine Order, all these patients were admitted marked "N.Y.D., N." (not yet diagnosed, ?nervous), and such cases are divisible into two general categories; the first and most serious is one of gradually acquired nervous breakdown, not referable to any specific explosion or to any accident such as being blown up or buried; it is obvious that any complete recovery from this condition, which may have taken two years' exposure to develop, is not to be expected in the course of a few weeks; it is discussed in this paper under the name of Chronic War-Neurasthenia. The second category includes cases rapidly induced by some of the accidents of warfare just mentioned, and differs from the former only in degree and not in kind; being rapidly induced it is much more rapidly cured, and it often has superadded to it hysterical symptoms such as mutism and paralysis, which are easily treated, although great sources of distress as long as they persist. These are the cases which are called Shell-shock, a most ill-advised but now established term. Shell-shock may, of course, occur with neurasthenia, or may form its starting point, or may first draw attention to it. I prefer to regard it as a state of Acute War-Neurasthenia.

The distinction quoted above is of little clinical interest, but was required by the higher authorities for disciplinary reasons, and at this time shell-shock was considered as a wound, and the distinction is preserved in all records, but in this paper the neuroses of war are considered as far as possible as a whole. Taking the distinction for what it is worth, there were among 1,300 cases, 612 of acute, 334 of chronic or established neurasthenia and 354 which were complicated with some form of organic disease such as bronchitis, rheumatism, pyrexia, or other disorders, requiring transfer to other departments or hospitals. The number of cases of organic disease of the central nervous system was exceedingly small, and was quite neglibible as having any bearing on the question of "N.Y.D., N."

With this material an attempt is here made to describe the neurosis of war as a clinical condition under the established headings of Definition, Etiology, Pathology, Symptoms, Physical Signs, Diagnosis, Prognosis, and Treatment.

If the popular word "nerve" correctly expresses the capacity to resist emotional strain, then I think the word *neurasthenia*, "weakness of nerve" is an accurate one for the condition found when that capacity

is exhausted. But neurasthenia has other associations in most people's minds, and is frequently used in a derogatory sense, and if I have any point to make it is that there is nothing derogatory in neurasthenia honestly acquired on service. I have therefore prefixed the word "war" as a qualification, since "polemo-neurasthenia" would merely be precious, and would add yet another title to a list already too long.

Definition.

A state of fatigue of the central nervous system, without organic change, manifested by asthenia, loss of physical and emotional control and disturbance of visceral functions.

Etiology.

In general terms the cause is physical and emotional strain, and the condition is due to yielding thereto; and since yielding to any strain involves consideration of the character of the strain itself and the qualities of the thing strained, both have to be considered under exciting and predisposing causes.

Search for predisposing causes has been unprofitable; neither age nor occupation afford any guide.

Age.—The age-incidence in a thousand consecutive cases has been worked out as follows: the cases are divided into five yearly groups—18 to 22 (and under), 23 to 27, 28 to 32, 33 to 37, 38 to 42 (and over), with this result: first group, 381; second, 274; third, 184; fourth, 89; fifth, 72. This, of course, is of no value except when compared with the proportions of troops serving in these age-groups, and such figures are not available. At a guess, the two correspond fairly closely, though it may be doubted if 38 per cent., that is, more than one-third, of soldiers exposed to trench warfare are under 23 years of age. On the whole, it seems probable that the very young soldier is especially liable to nervous breakdown.

Occupation: This was classified in 500 cases with equally unprofitable results. The occupations are grouped as: *Indoor trades:* weavers, mill-hands, dyers, metal workers, &c. *Indoor callings:* clerks, draughtsmen, shopmen, servants, &c. *Outdoor trades:* bricklayers, painters, plasterers, &c. *Outdoor callings:* labourers, chauffeurs, carmen, police, &c., *Underground workers:* miners. And give the following results:—

Outdoor trades and callings, 231. Indoor trades and callings, 225. Miners, 44.

"Nerve," as estimated by the patient, has been tabulated in 300

cases, and very roughly grouped as "good" or "bad"; "good" being the men who had had no trouble with their nerves at home, who could manage horses or work on high ladders, and who took their amusements in such sports as boxing or football; "bad" are men who had only been able to follow the less exacting occupations in civil life, who played no rough games and had suffered from "nervous breakdown." Intermediate cases were put as near one group or the other as might be. Among these cases there was a small majority on the side of those with "good" nerve. There has been no opportunity of estimating how far the internal discipline of any given battalion modifies the incidence of neurasthenia among its members.

There is enormous difference in the resisting powers of individuals; some men come down in a few weeks, a few have lasted three years and a half, and all intermediate stages are to be found, but they appear to depend much more on a man's individual qualities than on his circumstances, that is, they are a matter of heredity rather than environment. It is, of course, what one would expect; if you start a dozen men level to run a mile race, they will finish in a string, according to their lasting powers; and so it is with their lasting powers for the strain of war. The type known to neurologists as "degenerate," which contains many epileptics in its ranks, with the physical characteristics of narrow palate and crowded teeth, simian hand and coarse skin, has been found frequently among these patients. I have come to regard a narrow palate as indicating a bad prognosis.

Exciting Causes.—These are the various physical and emotional stresses of war, and may be individually well borne but producing cumulative effect (chronic war-neurasthenia), or, if sufficiently sudden and violent, may produce immediate effect (acute war-neurasthenia); specimens of each are given from patients' histories.

Gradual onset (chronic type). The cases are arranged according to length of active service.

Case 1.—Sergeant H., aged 22; two years in France. Previous nerve good, played football for battalion, boxer; has done very well, done everything, lasted through Somme, wounded, promoted Sergeant, D.C.M. Suffering from general strain, no special explosion. For three months bad sleep, loss of concentration, worrying, losing flesh, irritable, avoids society, nothing to say, emotional, loss of self-confidence, formerly fond of risk.

Case 2.—Sapper L., aged 32; two years in France. Nervous in any danger, no games or sport. Twice in blown-up mine, much frightened but not injured. Since then nerve worse. Kept back at headquarters. No recent shock;

two weeks ago returned to line, under heavy bombardment, could not stand it.

Case 3.—Private M., aged 22, one year nine months in France. Nervous at night, otherwise nerve fair. Good in France, wound fifteen months ago, nerve unaffected, lasted well till recently, through the Somme, four times "over the top." Two weeks ago heavy shelling, shell dropped in next bay, alarmed, not knocked down or unconscious. Since then has lost control in heavy shelling, no true shock. Recently four days under fire, became shaky, running about trench, not knowing where to go.

Case 4.—Lance-Corporal K., aged 21; one year six months in France. Nerve never good at home, but good in France for nine months. One year ago "shell shock," away four months. Served as pioneer through winter; all right till one month ago when battalion went into line, served one day, became excessively nervous, trembling in any shell fire, with præcordial pain.

Case 5.—Sergeant S., aged 28; one year five months in France. Nerve good, footballer, regular soldier. Out November, 1914, wounded Neuve Chapelle; rejoined July, 1915; shocks Somme, 1916; rejoined April, 1917; nerve good till Somme, since then deteriorated. No good now, cannot stand any shelling. Five days ago, some hours' bombardment, not very heavy, not laid out by any shell, taken to aid-post.

Case 6.—Private McG., aged 31; one year two months in France. Previous nerve good, boxer, swimmer; good in France at first, no wounds; well till five months ago when next bay was blown in, stunned, unable to carry on; one month at transport, returned to line, sent down again three times. Shaky in all excitement, at first only under bombardment, now on the least provocation.

Case 7.—Gunner P., aged 34; one year one month in France. Previous nerve good; footballer, runner, cyclist; nerve good in France, no wounds or shock; deteriorating since the Somme, mate killed in dug-out he had just left. Six days ago battery shelled out, thrown across trench, dazed but not unconscious, could not carry on.

Case 8.—Pioneer L., aged 20; one year in France. Nerve good at first; nine months ago blown out of bed, billet destroyed, mate killed. Nervous in Somme, some shock, but did not leave unit, no recollection of what happened for a fortnight, record of some unusual behaviour: permanent light duty since. Shocked again later, memory imperfect. No shock this time. Reported sick frequently with pains in head, weakness in legs, tingling limbs and inability to open fingers.

Case 9.—Private Le G., aged 18; ten months in France. Previous nerve never very good, but good in France; buried six months ago, away six weeks. Domestic worry, father, mother and brother all killed, nerve not good since. Sent out of the line two or three times. No recent shock, has several times fainted on the march.

Case 10.—Private K., aged 21; nine months in France. Nerve poor at home, better than he expected in France. Gradual deterioration, no wounds or shock.

Case 11.—Sergeant M., aged 26; six months in France. Gradual deterioration, nerve poor after shelling but carried on at first; later sent down by officer for a month, returned to line, tremulous under fire, twice sent down.

Case 12.—Gunner M., aged 23; three months in France. Nerve poor. No good on guns, upset by our own fire; no shock, reported sick with nerves and loss of sleep.

These cases illustrate well the different lasting power of different men, from fine soldiers like No. 1, to such "military misfits" as No. 12, and the varying period which they take to arrive at the same conditions.

Sudden onset (acute type).

Case 13.—Knocked out by heavy shell, unconscious probably an hour. Shaky, pains in head, emotional, laughing and crying.

Case 14.—Buried, not unconscious, lost self-control; emotional, shaky, unable to carry on.

Case 15.—In gun-pit, direct hit on gun; thrown about ten feet on to ammunition, stunned a few minutes, able to get out alone, shaky, not crying, very frightened.

Case 16.—Partly buried by trench-mortar shell, knocked out, could not carry on, trembling, not emotional.

Case 17—Asleep in dug-out, knocked over, not unconscious, could walk down, became unsteady, shaky, crying.

Case 18.—In dug-out which was wrecked, unconscious half an hour, could walk down with help, unable to carry on, shaky, head aching, not emotional.

Case 19.—Knocked out by trench-mortar shell, unconscious more than an hour; later, mute for three days, stammering two days.

Case 20.—Shell entered dug-out, unconscious an hour, shaky, walked down with help, lost speech, unable to carry on, emotional.

Case 21.—Caught up in wire, blown up, unconscious an hour, could not stand, giddy, dazed, emotional.

Case 22.—Knocked out, buried, stunned a short time, dazed, imperfect memory, shaky and crying.

Case 23.—Buried, stunned a few minutes, deaf, ringing in head, shaky, not emotional, could speak, unable to carry on, could walk down.

Case 24.—Knocked out, unconscious a short time, buried, could not walk, trembling and emotional.

The similarity of these histories is remarkable, and compared with those of the cases of the chronic type, shows that the only differences between the two are due to the rate of their onset, whether suddenly or gradually induced. Out of 1,200 cases 822 were of rapid and 378 of gradual onset, roughly 70 per cent. and 30 per cent., rather more than 2 to 1.

The exciting causes of war-neurasthenia then are *physical fatigue*, *physical violence*, such as being blown up and buried by shell explosions, and *emotional strain* such as continued alarm, the loss of comrades, other distressing sights and sounds, along with the continuous effort of self-control under impulses to seek safety.

Pathology.

No information was obtained as to the morbid anatomy of the condition. Three deaths occurred in the 4,700 cases of this series, two from cerebrospinal meningitis, and one from broncho-pneumonia. Post-mortem examinations were made in each case, but the state of the brain and spinal cord and other organs threw no light on the pathology of war-neurasthenia.

In cases of sudden onset such as that described in Case 15, in which a man was said to have been thrown 10 ft. across a gun-pit on to a heap of ammunition, it is clear that any degree of physical injury may occur. I have seen cases of fractured base of the skull, and fracture-dislocation of the spine. I have heard of a case in which the corpus callosum was found to be torn across without external injury, and many minor lesions occur, such as hæmorrhage into the conjunctiva or the membrana tympani, and punctate lenticular opacities said to be due to physical violence. I have records of seven cases of epistaxis, and one of hæmoptysis. All such things, however, are in my opinion complications of the condition. Punctate cerebral hæmorrhages have been described by several eminent neuro-pathologists, but if these were the rule, one would, I think, more frequently find evidence of organic injury, such as lesions of cranial nerves or about the red nucleus, or changes in the reflexes of organic type. Judged by the old-fashioned standard of the reflexes the condition is essentially one of functional disease of the nervous system, by which I mean a condition in which all nerve cells and fibres are structurally intact. These cases, therefore, provide no pathological facts. I give later an hypothesis as to a possible physiological perversion which would explain some of the symptons.

Symptoms.

A severe case, with sudden onset, without hysterical symptoms, seen a day or two after the event, presents an appearance much as follows:—

The general appearance of the patient is apathetic, while in quiet surroundings his face is rather anxious, but has not much expression; sometimes the eyes are widely opened in a stare. He starts violently

at any loud noise, and may duck suddenly and excessively on passing under cover, as for instance through a tent door. On questioning he will volunteer very little; he is often very slow to understand, he hears the words but there is some delay before he takes in their meaning; he is generally quite frank in his answers, which are nearly always corroborated by statements from his unit, and prove to be rather under than over estimates of the fact. He is frequently emotional, and breaks down into tears under examination. His general aspect is one of fatigue; he is slouching in movement, stands with rounded shoulders, bowed head and slack knees. The strength he exerts is below what is to be expected from his development, all movements are made with reluctance. He complains as a rule of headache, and often of præcordial pain; he is dizzy on rising. The pulse-rate is often rapid. The appetite is poor, and constipation is frequent. Micturition is often precipitate. Tremors are common, often increased on intended movement; the deep reflexes are generally very brisk without increase in tone. The pupils are sometimes widely dilated. Sleep is generally delayed and broken. The course is on the whole towards recovery, at any rate in first attacks, and proceeds at a speed which varies with the severity and prolongation of the strain, and the natural recuperative power of the patient. Recovery is not as a rule uniform, but is broken by a series of recurrences of symptoms, becoming as a rule progressively slighter and at greater intervals. The increasing smartness displayed by patients at sick parade is very striking, and there is in many cases a point at which men definitely turn the corner; for some days or weeks there is a reluctance to admit improvement, associated, as I believe, with a feeling that they could not face service conditions; but there comes a day when a man stands up perfectly straight, clicks his heels and salutes smartly, and when asked how he is replies, "I feel champion, sir."

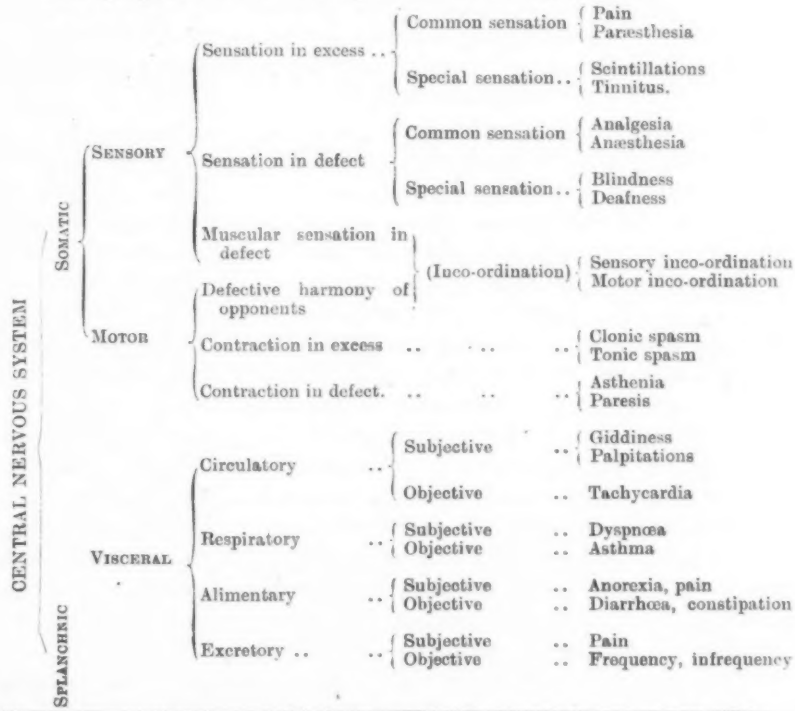
Headache	736	57	per cent.
Disturbed sleep	515	40	,,
Inco-ordination	374	29	,,
Pain, other than headache ..	303	23	,,
Asthenia	266	20	,,
Alimentary symptoms ..	251	19	,,
Cardio-vascular symptoms ..	237	18	,,
Urinary symptoms	204	15	,,
Psychical symptoms ..	196	15	,,
Aural symptoms	191	14	,,
Respiratory symptoms ..	104	8	,,
Ocular symptoms	92	7	,,
Mutism	85	6	,,
Paræsthesia	78	6	,,
Paresis	41	3	,,
Anæsthesia	37	3	,,

I have made a numerical estimate of the symptoms complained of in these 1,300 cases, which is given above; some of them are to be regarded as complications, but they are all grouped together in a single series. They are compiled from the histories taken on the admission of patients, thus avoiding symptoms due to suggestion from and mimicry of comrades. The subjective ones are only those volunteered by patients.

On the preceding page is the incidence of the symptoms in these cases in tabular form.

Having adopted the view that war-neurasthenia is primarily a functional disease of the central nervous system, I take the directly referable symptoms of the disorder to be disturbances of the functions of sensation, movement and co-ordination; but visceral disturbances require separate consideration. Properly, in my opinion, hysteria is not of the essence of the condition and should be regarded as a complication, but the two are so closely associated that separation is impracticable.

The symptoms are divided up on the following scheme:—



Also PSYCHICAL SYMPTOMS and disturbances of SLEEP.

It will be observed that the commonest symptoms are those of common sensation in excess, disturbance of sleep, and disturbance of co-ordination, that pure motor symptoms, with the exception of asthenia, are rare, that visceral disturbances and also psychical symptoms are common though not invariable, and that disturbance of the special senses is infrequent.

The chief of these may be considered rather more in detail. Notes were made only when the patients volunteered information about their condition, it being desirable to avoid suggestion. Had more inquiry been made, the numbers of all symptoms would doubtless have been larger, but I expect the proportions would have been maintained.

Disturbances of Sensation.

Pain.—Headache, 736 cases.

Site.—Described as frontal in 65 cases, occipital in 55, vertical 17, unilateral 15, temporal 13, eye 8, referred to site of old wound 9.

Time.—Worst in early morning 16, at night 13.

Induced by change of position rising or stooping 17, by noise 16.

Character.—Shooting 17, throbbing 10, pressure 10, neuralgia 5, and occasionally described as "bursting," "top lifts up and down," "rattles," "splitting," "knocking," "in a vice."

I have not gathered the impression that this headache, so commonly complained of, is in fact usually very severe. It is often stated to be present in men who have no appearance of being in serious pain, and when pressed by such questions as, "But you are surely not in very severe pain now?" will admit that they are not, but say that they have recently been so. Like all purely subjective symptoms, it is of course liable to exaggeration and also simulation, and my own impression of it is that it is real, not as a rule severe, but always ill-borne. Occasionally one is convinced of its severity.

In treatment I have found drugs of very little value; the tar products, aspirin, phenacetin, and the like do little good. Physical applications are the best; I have had good results from hot-water bottles and fomentations for occipital pain, and ice-bags and evaporating lotions for frontal pain; blisters to painful spots have several times given relief, and there sometimes is improvement when pillows are packed so as to raise the head at night; instructions should always be given to change the position slowly.

Lumbar puncture was performed nineteen times for relief of pain

with the following results. The puncture was always made in the sitting position, without an anæsthetic. The pressure was never measured mechanically but evidently varied considerably; in some cases a well-maintained flow was obtained, while in others there were only a few drops. Several times, though the needle was apparently in the theca, no fluid was obtained.

Case 1, 15 c.c., no relief.
Case 2, 15 c.c., much improvement.
Case 3, 15 c.c., relief for one day, recurred.
Case 4, 15 c.c., much relief.
Case 5, 15 c.c., great and maintained improvement.
Case 6, 8 c.c., great relief, changed character.
Case 7, 7 c.c., great relief, changed character, recurred.
Case 8, 4 c.c., temporary improvement, recurred.
Case 9, 2 c.c., relief.
Case 10, 1 c.c., no improvement.
Case 11, a few drops, no improvement.
Case 12, a few drops, slight improvement.
Case 13, blank, relief, recurred.
Case 14, blank, relief, recurred.
Case 15, blank, relief, recurred.
Case 16, blank, some blood, great and maintained improvement.

In three cases the needle did not appear to have reached the canal, but in two of these the patients described themselves as better.

The precise value of lumbar puncture is not very clear from this; but as a rule where headache is persistent and really appears severe, plenty of fluid can be withdrawn and this is often followed by striking relief.

Pain, other than headache.—Recorded in 303 cases; where particulars are given they are as follows: They are chiefly referred to the back, shoulders, loins and neck (61 cases); they are generally described as "aching" without more detail, sometimes "like a red-hot needle sticking in my back."

Pain in the chest, generally definitely præcordial, comes next (47 cases); this is variously described as a "grip on my heart," "catching," "shooting," or added to a sense of heart-stopping. This is referred to again under the Circulatory System.

Pain in the legs is noted thirteen times, in the limbs four times, and "all over" twice; definite cases of trench fever (recurrent pyrexia and shin pains) are not included in this list, but these cases probably belong properly to that category.

Pain in the abdomen is complained of six times, and pain in an old wound, or the site of an old pleurisy, occurs five times (*cf.* headache, referred to the site of an old wound).

I have found here again that physical remedies are most effective, and that blisters to the chest are the most satisfactory treatment for præcordial pain.

Hyperæsthesia though occasionally met with was never investigated.

Paræsthesia.—Details of paræsthesia were not sought after.

Those noted are chiefly numbness and cramps in the extremities. A curious abdominal sensation is often complained of, and is nearly always described in the same way—"My stomach turns over and over." General malaise is not infrequent, and globus hystericus was occasionally noted.

Anæsthesia and analgesia were not systematically looked for, but under this heading I have included cases of stupor and of hysterical fits. There are notes on thirty-seven cases of anæsthesia. Anæsthesia in these cases is an hysterical manifestation, and is a condition in which stimuli of ordinary intensity, though received by the afferent nerves, are not realized, or at any rate not interpreted, by the centre of consciousness. This applies equally to common sensation and to the special senses. The depth of anæsthesia varies, but if sufficiently powerful stimulus can be applied it will be felt, and once anything is felt stimuli of ordinary intensity can soon be felt likewise. This is the general rule: cases occur in which no stimulus which can be applied without risk of injury can be felt.

The most pronounced cases met with are those of stupor, of which I have notes on nine. The majority were roused without difficulty; one man remained stuporose for three or four days; he could be induced to take food, but otherwise lay in bed taking no apparent interest in his surroundings. The commonest condition is that of a man who will make no spontaneous action, and has to be carried on a stretcher; he generally has his eyes open, but does not understand questions; he may ask for a smoke or a drink, or call out for a comrade. In milder cases he can be induced to get up and move about in an automatic manner; in this state he will walk out of a tent without his clothes, unless checked. At times a patient cannot be roused at a single session, but after a few hours in bed, or perhaps the following morning, wakes up quite rational. The following cases illustrate degrees of the conditions:—

Case 25.—Rifleman R., carried to inspection tent on stretcher. Quite unaware of surroundings; could copy movements. Induced to sit up. A dinner bell was rung loudly behind him, and after a few seconds he began to flinch and hear. Soon after this he became aware of where he was, and in a few minutes could read and identify objects. He then gave his name in a whisper, rapidly recovered phonation, and became quite rational. He stated that his first consciousness came with hearing the bell; his memory gradually returned.

Case 26.—Private G. Could neither hear nor read, but was able to eat and to dress himself. Faradization with wire brush, strong current not felt at all for several minutes, but afterwards it became painful and he cried a little, and said, "It hurts." Flinched at sound of a loud bell. Copied movements. Next day consciousness returned spontaneously, he could hear but not speak; speech was restored in a few minutes, but with a stammer, which improved but was never quite lost while in hospital. His memory was quite clear up to the time of his shock.

Case 27.—Lance-Corporal H., stated to have been alternately laughing and violent at the regimental aid-post. On admission he could understand signs a little, but could neither hear nor read. Roused a little by faradization, and induced to stand.

Next day there was some improvement: he could walk, but would walk out of the tent half-dressed; he flinched at the bell. A few hours later consciousness returned; he could hear one's voice at about four inches, but could not speak; speech was restored in about an hour, but was very slow and hesitating. He was quite intelligent, but had persistent difficulty in finding the right word, either spoken or written; this was probably partly because, though an Englishman, he had lived chiefly in France, and did not know English very well.

Case 28.—Rifleman N., admitted unconscious, gesticulating wildly and talking gibberish. Under strong faradization recovered consciousness gradually, was induced to stand, answered his name, gave name and unit, and wrote his address, reversing several letters.

There were two other cases of general anæsthesia without unconsciousness, one marked case of hemianæsthesia and hemianalgesia, one of anæsthesia of the legs associated with paresis, which recovered under faradization, but relapsed.

There were three cases of violent hysterical fits, aborted in the usual way by cutaneous stimulation.

Loss of sense of deep pressure on muscles is considered later under "inco-ordination."

Disturbances of special senses, both excess and defect, are considered together, along with the accessory symptoms such as squint, which are generally referred to specialists.

Ocular symptoms : Ninety-two cases.

Change in visual sensation.—No cases of functional blindness occurred, but vision was "blurred" in twenty-eight cases, sometimes especially on rising, and probably associated with the dizziness on rising previously noted.

Inability to read because "print all runs together" is a common complaint, and failure to accommodate for near vision, the effort failing below 12 or even 18 inches, was many times observed by Captain Evans. I have noted the fact twelve times. Associated with this are diplopia, squint and imperfect outward movement of the eyes noted ten times, in one case a definite convergent squint with spasm of accommodation was relieved by atropine. Another case of headache referred to the same cause was relieved in the same way. There are thus cases both of spasm and of failure of accommodation. Many subjective phenomena are complained of, scintillations or specks thirteen times, chromatic abnormalities eight times. The following are specimens: "Sees objects moving at night, different colours," "colours floating before the eyes," "things go black," "everything went blue," "everything goes white," and also "sees rings which get larger and smaller," "objects recede on trying to focus," "everything seems bigger." There is occasional photophobia.

In nine cases it was noted that the pupils were very wide, but reacted to strong light. In thirteen cases the eyes were very widely opened, showing the whole cornea in a stare, and sometimes showing von Graefe's sign of delayed descent of the upper lid on looking down. I have never seen any true exophthalmos in these cases, the eyelids merely being held very widely open. Lenticular opacities were observed three times, and once attention was drawn to the condition of an old amblyopic eye, which had previously given no trouble; this is probably comparable to the reference of pain to the site of a previous wound.

A point several times observed is that patients obstinately refuse to look at the examining medical officer. Hæmorrhages into the conjunctiva were occasionally seen.

Aural symptoms : 191 cases.

The symptoms complained of are deafness, deaf-mutism, and tinnitus.

Deafness may be functional, organic, or functional on an organic basis. In aural disease, perhaps more than any other, is a previous or latent injury likely to be aroused.

There is pure functional deafness, with or without mutism; it is an anæsthesia, and neither bone-conducted nor air-conducted vibrations

are appreciated. In many instances, as described under Anæsthesia, a sound, if sufficiently loud, can be heard, such as a loud bell. Words shouted through a stethoscope can often be heard, and once heard, ordinary hearing may be quickly restored. In the course of a single session a patient, who can hear nothing at all at first, may be able to hear words spoken at the ordinary conversational pitch, say in half an hour's time. Guard must be kept against the psychical condition in which words can be heard and repeated but convey no meaning. Some cases are very resistant, many will flinch when words are shouted down the stethoscope, who are not conscious of hearing any sound.

Deafness may also be organic, all degrees of injury to the tympanum and ossicles being met with. Acute otorrhœa is, of course, very common in youth as a sequel of measles, scarlet fever and respiratory catarrh, often becoming chronic or latent. Such cases are frequently much aggravated both by exposure to shell fire, and by the imperfect facilities for cleanliness available on service.

Tinnitus is still commoner than deafness, and is described variously as "ringing," "thumping," "bell ringing," "hissing," "throbbing," "like motor lorries," "drumming," and so on.

The prognosis for hearing is good in functional cases. I have the following notes:—

Case 29.—Deafness, recovered.

Case 30.—Deaf-mute, cured at once.

Case 31.—Deaf-mute, induced to speak, hearing recovered in a week.

Case 32.—Hearing lost, gradually recovered.

Case 33.—First day, could not hear own voice; second day, could hear noises but not distinguish sounds; third day, could hear fairly. This patient relapsed.

Case 34.—Deaf-mute, speech restored, and with it some hearing, but this was completely lost later, although he flinched on shouting down the stethoscope. There was considerable organic damage to the drum.

The most effectual treatment, in my experience, is daily re-education with bells, stethoscope and so on.

Disturbance of other special senses has rarely been met with; there was one case of parosmia, the man being aware of a persistent smell of poison gas, and one case of the loss of the functions of taste and smell, in a grocer with a cultivated taste for butter.

Disturbance of co-ordination.—The faculty of muscular movement is useless for the purpose of human activity without the faculty of precision of movement, which is attained by the co-ordination of muscular con-

tractions. Co-ordination has two aspects—sensory and motor. For any movement of precision it is necessary to be aware of the position in space of the limbs involved, which varies as different joints are moved, and this depends on the integrity of the sensory apparatus, and if that is out of order precision cannot be attained. Motor co-ordination is the harmonious action of opposing groups of muscles which control the degree of movement of individual joints. Such a movement of precision as that of bringing the hand from the side to the mouth requires flexion of the elbow, by contraction of the biceps and brachialis anticus, but flexion must be stopped at a given point and this depends on the action of the antagonist of these muscles, namely, the triceps, which acts as a brake and checks the movement of flexion at exactly the right moment.

Both sensory and motor co-ordination are disturbed in organic diseases which involve special parts of the central nervous system concerned in these functions. Sensory co-ordination is disturbed in tabes dorsalis, the tabetic gait being due to loss of sense of position of the limbs, so that the feet are thrown unduly high in the air, because the sensory impulse which should indicate their position is absent. Motor inco-ordination is present in disseminated sclerosis when the red nucleus or the tracts communicating with it are disordered. When this occurs, muscles and their opponents do not act in harmony, but alternately, producing tremors, and precision of movement is thus lost.

Both sensory and motor co-ordination may be functionally disturbed after shock.

Sensory inco-ordination.—Tabes dorsalis is the typical organic condition which produces sensory inco-ordination, and the general assumption is that in tabes those parts of the cord are first affected along which impulses from muscles and joints are conveyed. Consequently one finds the tabetic gait, and the common phenomenon of missing the nose with the finger with the eyes closed, and increase of unsteadiness in standing on closing the eyes. In addition to this there is astereognosis, inability to describe the position of limb without seeing it; loss of muscle pressure sense, when the calf or other muscular mass can be squeezed forcibly without pain; and also the loss of tone and abolition of the deep reflexes. Many of the phenomena in this list, namely, inco-ordinate gait, Romberg's sign, astereognosis and loss of muscle pressure sense may occur after shock, though not as a rule all together. Confusion with tabes may easily arise: the following case occurred early in my experience:—

Case 34.—An officer, aged 41, three months in Ypres Salient, nerve unaffected. Eight days before admission was knocked out by a shell, unconscious

a short time, then carried on for twenty-four hours. Suffered from severe headache and sense of numbness over occiput, difficulty in walking, great weakness and loss of control of legs, walked as if drunk. Passed no urine for two days, bowels not open for four days.

On examination: Pupils sluggish but reacted to light. Transient squint. Great inco-ordination of hands of sensory type, missed his nose by several inches with the eyes shut, inco-ordination of legs, unsteadiness in standing much increased by closing eyes, gait inco-ordinate; sphincters, delay. Deep reflexes, knee-jerks increased, ankle-jerk present.

The combination of sensory inco-ordination of arms and legs, transient squint, and delayed relaxation of sphincters, occurring in a man of 41 after prolonged exposure to severe campaigning is strongly suggestive of tabes, which was my diagnosis, but all symptoms cleared up after two or three days' rest.

I have found sensory inco-ordination, as shown by the finger-nose test, in fifteen cases, loss of position sense in eleven cases and loss of muscle pressure sense, in the absence of any particular anæsthesia of the skin, in six cases. A reeling gait, in which, as a rule, the legs are crossed, is sometimes found, an hysterical Romberg's sign may be found, and such ordinary functional symptoms as astasia-abasia and retrogression are not uncommon.

Motor inco-ordination.—This is manifested by tremors, by which I mean the alternate rapid action of antagonistic muscle groups about any given joint. The most frequently observed being alternate slight pronation and supination of the wrist producing the shaky hand, and alternate slight rotation of the neck to right and left, producing the shaky head.

The tremors may be general or localized to any part; they are most noticeable and most inconvenient in the hands, partly from the long leverage given to any movement of the hand, which originates at the elbow, and partly from the interference with necessary actions. The tremors are often of the "intention" type, which is generally applied to those found in disseminated sclerosis, the excursion being increased during any attempted movement of precision the nearer the movement is to completion.

All degrees of the condition are found, and it is much increased by any alarming occurrence, such as gun fire.

Stammering is one of the commonest forms of inco-ordination found in war-neurasthenia, naturally enough, perhaps, since speech is a faculty requiring the co-ordination of several distinct muscle groups. The respiratory muscles must be working smoothly, the muscles of the larynx must be accurately co-ordinated with them, to produce phona-

tion, and along with these must go the muscles of the tongue and lips on which articulation depends. The cortical centres for tongue, lips, and larynx are all very closely situated, and are, so to speak, evidently intended to act together, but, none the less, their several actions are often enough functionally disturbed.

Be that as it may, stammering is one of the commonest, and often one of the most intractable of disturbances of co-ordination. It is sometimes an aggravation of a pre-existing stammer, or a recurrence of a juvenile one.

It may be laryngeal, the larynx being kept closed and the breath not allowed to pass, which causes delay and struggle in starting speech, and sometimes the pitch is out of control, syllables are dwelt on unduly long, and the voice runs up the scale.

Sometimes it is chiefly articulatory, the patient failing to separate the lips for a "B," or the dorsum of the tongue and the palate for a "K," or else the letter can be made but is repeated an indefinite number of times.

Apart from actual stammer there is a peculiar speech which I have come to regard as characteristic of war-neurasthenia. It is low pitched, slow and monotonous, and it is "spaced," each syllable being given an equal value, with a pause between each; the sentiments conveyed are generally those of a depressed person, and are discouraging. "I—don't—feel—much—bet—ter—this—morn—ing," is the usual kind of remark. Stammering often occurs in patients previously mute, and often clears up completely in a day or two, sometimes as suddenly as the restoration of speech. In others there is gradual and maintained improvement, and this may be much assisted by treatment, which is of value in direct proportion to the trouble taken by the patient. Many cases of stammer are very resistant, and may continue unchanged for several months; beyond this I have no experience.

With regard to response to treatment, disturbances of co-ordination are far more intractable than those of paresis, which is remarkable since many cases of paretic spasm are more easily understood as cases of inco-ordination than otherwise.

Disturbances of motor system.—Contraction in excess, tonic and clonic spasm.

Both of these were curiously rare, only four cases of spasmodic contraction are recorded; all were cases of rigidity of one or both legs. Such cases have a good deal in common with inco-ordination since the rigidity is generally due to failure to relax the muscles

which oppose the desired movement. The leg is held rigidly extended at hip and knee, generally extended and inverted at the ankle. Passive movements are fairly strongly resisted, and on a voluntary attempt at flexion the extensors harden and the spasm becomes slightly clonic. When walking is possible, the leg is held stiffly and circumducted. The condition differs only from that seen in civil practice in that it is recently acquired, and proportionally easily cured.

Clonic spasm, tic.—This again is uncommon. I have notes on only thirty cases, and these too differ only from those seen in civil life in their shorter duration; no particular treatment is required, in a large number of cases they subside in a few days. I regard it as ground for rather bad general prognosis; a large proportion of them occurred in severe cases, who were sent away quickly to base hospitals, very few were found fit to return to duty, and some of these were soon re-admitted.

Many different parts of the body were involved, head, face, eyelid, arm, shoulder, respiratory muscles, limbs, and "general." Seventeen cases cleared up, as to their tics, in a week or two, while several of the remainder were evacuated in a few days as severe cases. This is a much higher percentage of recovery than occurs in established tics treated in civil practice.

Contraction in defect.—Asthenia and paresis are much commoner than contraction or tic. Some degree of asthenia is the rule, it is part of the exhaustion following on the strain which is the exciting cause of the condition. I have found it evident enough to receive notice in 266 cases.

The commonest condition is simply one of objective weakness, inability to offer resistance to any strain on the limbs, as revealed by ordinary tests such as holding the fist up to the shoulder and resisting it being pulled away. When the weakness is not extreme, it is often obviously below what would be expected from the patient's physical development. Subjective asthenia, a feeling of weakness, is occasionally complained of, but an evident reluctance to make any effort and ill-maintenance of effort are the rule.

Paresis.—This occurred in forty cases, generally in the legs; paresis of the larynx with mutism is considered separately.

Paresis of the legs is generally an extreme condition of the reluctance noted above under "Asthenia." It is rarely complete, it does not often occur that a man is quite unable to move his limbs at all. He is, however, convinced that he has lost their use, as far as walking is concerned, and makes no effort.

The great majority are rapidly cured, most will walk back to their tents after one session of treatment. I remember none which were not at least able to stand. In some cases progress is slow and some defect in gait may persist for many weeks.

In a severe case the patient has to be carried to the examiner's tent. When lying in bed and asked to raise his legs, he can only lift up his foot a few inches. After some passive movements of the limb this improves, especially with encouragement. Asked to get up he generally protests his inability, and may have to be helped to a sitting position on the edge of the bed, and on to his feet. He lets his hips, knees and ankles give way, and subsides on the floor. With encouragement and insistence and some help he can generally be got on to his feet, but stands very unsteadily and clutches at support. It is commonly said that such patients only "fall soft" and do not hurt themselves, but I have seen some very heavy falls. When a patient can once maintain his balance the worst is over; with assistance and encouragement he can be induced to shuffle a few steps, provided you assure him that you will prevent him from falling. Care is required that not too much is done at any one session. If over-fatigued, patients are easily put back.

The commonest paretic symptoms are mutism and aphonia; there were eighty-five in my series. They are in nearly every case rapidly cured, a great many recover spontaneously, and the great majority in one session of treatment. There were four really resistant cases who took several days or weeks to cure, the delay was generally due to error in treatment, they were difficult cases which were undertaken by myself at the end of a heavy day's work, when I was fatigued and irritable, and became obliged to give up the treatment because I found myself losing my temper. If failure is once made, the difficulty of ultimate success is much increased.

Sometimes whispered speech is restored, but phonation delayed, and often the voice is out of control, running too high in the scale for some time. Stammering is common as a sequel, sometimes so bad that a patient is practically dumb. There were several cases which were rather aphasia than mutism, the patient learning to phonate and being able to repeat words, but unable to make any use of them. About one in eight of the mutes were also deaf, and if attention was given to the mutism by means of written instructions it was found that the hearing improved with the recovery of speech. As in cases of stupor a very partial success at any session may be followed by great improvement, next day. In the great majority of cases speech was restored in a single

session of treatment, mutism is in fact one of the easiest symptoms to cure, and such cure has often something of the dramatic about it, but any claim to special merit put forward for any system of treatment based on its success in treating mutism should be heavily discounted.

Visceral Symptoms.

Disorders of the great visceral systems are considered on the assumption that they are functional disturbances of the splanchnic division of the central nervous system. The arrangement is merely one of convenience, and is not intended to imply that any research work has been done to demonstrate any functional derangement of the heart or bladder which is strictly comparable to functional disorder of muscular movement or cutaneous sensation.

Circulatory symptoms.—237 cases.

These are considered under the headings of Subjective and Objective Symptoms.

The subjective symptoms are those most commonly found, and are chiefly vertigo, described variously as going "giddy," "dizzy" or "mazy"; præcordial pain, palpitations and dyspnœa. The vertigo is frequently worse on rising, and is occasionally severe enough to cause falling and even syncope. Præcordial pain, as noted under pain, is described as "shooting," "catching" or "a grip on the heart," it is generally localized about the left border of the heart.

Dyspnœa, doubtless often cardiac in origin, is referred to under the respiratory system. Subjective palpitation is occasionally noted.

The objective symptoms are tachycardia, cold, sweaty hands, cyanosis and blanching of the extremities.

Tachycardia is often very considerable; in my experience, it is not very often an initial symptom, but is developed in the course of a week or two. It is remarkable, since in this campaign, with all men inoculated against typhoid infection, the pulse rate is on the whole slow.

In war-neurasthenia it is generally high, but I make no claim to a systematic study of pulse rates. The following were noted:—

Case 35.—Rest, 128; short exercise, 168.

Case 36.—Exercise (walking in tent, perhaps 10 yds. ; after five minutes' rest, 130.

Cases of pulse rate of 120 while merely attending an ordinary sick parade were very common.

An objective symptom, less frequently met with, but sometimes very

striking, is a condition in which the hands and feet are cold, sweating and cyanosed. The typical picture of a patient suffering from war-neurasthenia, in whom the circulatory system is affected, is that of a generally depressed person, listless and uninterested, with a slightly cyanosed face, much cyanosis of the hands, which are a little swollen, chilly and moist, and a pulse rate on standing of about 120 per minute.

Several times a condition indistinguishable from Raynaud's disease has been found, in which the fingers and toes are quite blanched, cold and numb, occurring in patients who are not subject to anything of the kind. I have notes of eight such cases. They all improved at once with the wearing of woollen gloves, and cleared up in a few days; they appear therefore to be distinct from the cyanotic condition previously mentioned.

Respiratory symptoms.—104 cases.

The only respiratory symptom complained of is *dyspnœa*, either subjective or objective, doubtless it is sometimes cardiac in origin.

Subjectively it varies from a general complaint of shortness of breath on exertion or in excitement to that of definite attacks of asthma, often nocturnal, and associated with sensations described as "inability to speak," "gasping for breath," "wakes smothering at night," and an impulse to open the collar.

There is sometimes very striking dyspnœa, kept up for very long periods of some days, at any rate during waking hours, though I have no record of the condition during sleep.

In one case the general appearance of the patient strongly simulated that found in pneumonia, the breathing was counted at 120 per minute but with a pulse rate of 100. While under examination an aeroplane passed over the tent and distracted the patient's attention for a few seconds, during which the respirations fell to the ordinary rate. The dyspnœa gradually subsided and breathing was normal in three days. In another case respirations were counted at 80 and the pulse at 64 per minute.

Alimentary symptoms.—251 cases.

The alimentary system is frequently out of order in war-neurasthenia. The commonest subjective symptom is anorexia, and the commonest objective symptom is constipation. On the other hand, diarrhœa is not infrequent and it appears that the functions of the intestines may be upset both towards excess and defect.

Subjective symptoms: Appetite is frequently bad, there is general lack of inclination for food, "I don't care whether I have it or not," and

often a meal is left unfinished, appetite rapidly failing. Thirst is occasionally complained of, and often during any prolonged examination or treatment the patient will drink greedily. Hunger, quite abnormal desire for food, is recorded twice. Loss of taste is recorded three times.

Nausea and dyspeptic pain are both fairly frequent.

Objective symptoms: Retching and vomiting occurred in about 10 per cent. of the cases with alimentary symptoms. Constipation is the rule. I have records in 35 per cent. of cases; three days' duration is a common period, and patients state that their bowels have not been open for five or six days or even longer.

Diarrhœa is recorded in 15 per cent. of cases. Small outbreaks of diarrhœa were rather frequent in the camp, and a large proportion of these patients were always affected.

Genito-urinary symptoms.—204 cases.

The only subjective symptom is pain in association with micturition, and this does not appear to be severe. Sometimes there is desire to micturate but no result.

Objective symptoms. Frequency is the commonest; it is chiefly nocturnal. Precipitancy leading to incontinence was found sixteen times, and nocturnal incontinence twice; it occurs in persons who were subject to enuresis in childhood. Several patients were asked to record the number of times they passed water in a day, a record which cannot be regarded as sworn testimony, but is still of some value. Some of the results are as follows:—

Twenty-nine times in twenty-four hours, passing 75 oz., sp. gr. 1008; twenty-three times in twenty-four hours, six times at night. (Five, four or three times are frequent.)

Infrequency is also common. These are instances:—

No urine for two days; micturition once in five days (alleged), once in two days, once a day, once in thirty-six hours.

The amounts of urine passed in twenty-four hours are also very variable. The following are recorded: 125 oz., 110 oz., 92 oz., 75 oz. and also 25 oz. and 15 oz. The contrast between excess and defect of the function of secretion of urine is very striking. As a rule there was no abnormality detected in the urine, and specific gravities were as a rule within normal limits, thus:—

125 oz.,	sp. gr.	1020,	no abnormal	constituents.
25 oz.	,,	1012,	,,	,,
63 oz.	,,	1015,	,,	,,
110 oz.	,,	1018,	,,	,,

On one occasion sugar was found in the urine of a patient admitted stuporose; it disappeared on the following day.

Persistent seminal emissions were several times reported, and were generally rapidly reduced by the exhibition of iron and arsenic.

A few other symptoms were observed, which are not classified under any previous heading, of which the chief are free salivation and dribbling, sweating, and premature greyness of the hair and baldness; I have records of men going grey since the war began at the ages of 33, 29, 28 and 26, who were aware of no family tendency to this change.

Psychical Signs.—196 Cases.

The psychical symptoms observed in a general physical examination are only those of an obvious character, since systematic mental investigation is not carried out.

The chief thing complained of was lack of concentration, inability to read because the words mean nothing, and incapacity to write because the patient can find nothing to say; to write an ordinary letter to his own relatives is often an exhausting mental effort. One man was admitted a deaf mute, speech and hearing were restored in about half an hour, but he could only understand the simplest questions when spoken quite slowly; he said that he could hear words but that they conveyed no meaning to him.

Along with this, memory is often imperfect, though to a very variable degree. The following two cases showed positive amnesia:—

Case 37.—Lance-Corporal D., aged 20. Admitted March 14, 1917.

Appears to have no memory of events since playing cricket in August 1914. Has heard of Kitchener's Army, does not know of Lord Kitchener's death. Does not remember joining the Army. Surprised to hear he is in France.

March 15, 1917.—Remembers coming to France and being ill on board ship: the bombardment of Lowestoft; his Captain; the Esplanade Hotel; his mate being killed by an explosion; periscope, and seeing barbed wire.

March 19, 1917.—Remembers much more: Calais; farm-billets; marching order; dug-out; several men there, bombs falling and a man killed; had to bind up other men.

March 23, 1917.—Remembers enlisting September, 1914, 1/5th Beds., not taken to Gallipoli on account of nerves. Trained at Newmarket, Southwold and Holtham. Came to France January 1917, battalion seems to have been in rest till lately. Remembers going down communication trench, resting in dug-out till dark, and then going to bombing post in the dark; remembers bombs falling and some men being injured. Memory for immediate past very bad;

does not remember dinner to-day, and only recalls these events during conversation, otherwise mind is blank. Evacuated to base hospital.

Case 38.—Private N. Admitted March 13, 1917, semi-stuporose; said to have been unconscious for about a fortnight; no recollection of past events.

March 15, 1917.—Still only partly conscious, obeyed instructions in unintelligent manner, but improved much in intelligence during conversation. Remembers saying good-bye to his wife and child, but no recent memories.

March 19, 1917.—Remembers seeing his brother-in-law in uniform, who wanted him to join up, but he would not; also being a collier employed in an Admiralty pit, and so in a starred occupation, he could not do so: no knowledge of France. This man showed no further improvement before evacuation to a base hospital.

These cases are exceptional; as a rule amnesia does not amount to more than a blankness of mind about the occasion of the shock, and occasional lapses of memory, closely associated with inattention and lack of power to concentrate. Varieties of the condition are periods in which a patient "loses himself"; one man lost his sense of direction. Again attention to ordinary affairs may be interfered with by the fact that they cannot get their minds off the distressing circumstances they have been through, and this again leads sometimes to extreme depression, a sense of impending calamity, and a feeling "like going to certain death."

Loss of emotional control, exhibited chiefly by frequent crying, often occurs. Nervousness, alarm, much increased by anything recalling terrible memories such as gunfire, is the rule; sometimes it leads to complete loss of self-control and at times to impulses to murder and suicide, though no case of attempted violence has occurred in this series. Hallucinations are occasionally complained of: "sees shells bursting as he lies in bed," "unearthly things speaking in my head." One or two cases of acute hysterical mania have occurred.

Next to headache, bad sleep is the commonest symptom complained of. I have it recorded in 515 cases. Like headache, it does not bear investigation; when put under observation it is found that most patients sleep well after midnight. Sleep is, however, both delayed and broken, and disturbed by horrible dreams. Talking in sleep is often reported by others. Sleep is often said to be prevented by headache, and may be disturbed by hallucinations of sight, hearing and touch, and a feeling that the bed is on a see-saw. The following phrasing of complaints have been noted, "wakes not knowing where he is," "wakes shouting and cursing," "wakes trying to find something," "broken by a vision of

his pal," "kept awake by worry." Somnambulism is rare. Occasionally (6 cases) patients complain of being unusually sleepy. It has to be remembered that these men are very listless and inclined to take every opportunity of sleeping by day.

Physical Signs.

Physical signs.—War-neurasthenia being essentially a functional condition, there are no changes in the shape, position or structure of the organs, and therefore there are no physical signs of the condition. There are, however, one or two points which may conveniently be referred to under this heading.

Tachycardia and the cyanosis, chilliness, and sweating of the extremities which occur in some cases of cardiovascular disturbance have been noted.

In the examination of the central nervous system some points about the reflexes require notice. No change was observed in the superficial reflexes, but the pupil was frequently found unusually wide, though it reacted to light. Deep reflexes showed an exaggeration of the variation often found according to the weather. This series of notes was begun in the winter, and throughout the cold weather the deep reflexes were found as a rule to be very brisk, in the usual functional way without any increase in tone; but during the hot weather of the summer the reverse occurred and it was often unusually difficult to obtain any deep reflex at all.

The sphincters, as noted under the alimentary and genito-urinary symptoms, are often disturbed both towards precipitancy and delay.

Diagnosis, Prognosis, Treatment.

Diagnosis.—The possibilities to be excluded are the following :—

(1) *Organic disease of the nervous system* and *insanity* which between them amounted to about 5 per cent. of my series, and included hemiplegia, paraplegia from fracture-dislocation of the spine, disseminated sclerosis, tabes dorsalis, traumatic peripheral nerve injuries, meningitis, peripheral neuritis, epilepsy, dementia paralytica, dementia præcox, mental defect and feeblemindedness.

My thanks are due to Captain Evans, who has experience both as an alienist and with the feebleminded, for his help in these cases.

(2) *Intercurrent disease,* of which trench fever (recurrent pyrexia and shin pains) is perhaps the commonest. It is an infection very widely

spread among troops, and one guesses that shock and strain predispose to recurrence in persons once infected. The pains are not uncommon without actual pyrexia, and it is not unlikely that trench fever accounts for a good deal of the pain complained of in war-neurasthenia.

(3) *Malingering.*—This, in the sense of deliberate simulation of symptoms, is in my opinion very rare. Exaggeration of course is common enough, and no doubt the purely subjective symptoms, such as headache, are often worked for a good deal more than they are worth. There is no guide that I know of beyond reports of different behaviour when a man is at sick parade and at other times. Against it all one has to put the fact of the enormous strain to which a man knows he is going back, after having previously broken down under it, and I am convinced that many men are speaking no more than the truth when they say that they do not feel fit to rejoin, although they would doubtless be fit enough for many forms of employment, provided they did not involve further exposure to conditions of warfare. But the large proportion of patients who are returned to duty without any difficulty indicates the absence of any real malingering.

(4) *Dislike of military service.*—This is a condition in many ways distinct from malingering, and I can only judge of it from hearsay. It is that of men who become distressed and alarmed in action, and on the first opportunity report sick in hopes of being sent out of the line, although they have undergone no real shock, and have not been sufficiently exposed to acquire war-neurasthenia. The sequel depends entirely on the battalion medical officer, for if a man finds that by making fuss enough he can get sent out of the line, he will make the first instance a precedent, as soon as he is returned to duty, and his career as a fighting soldier may be considered closed. Nothing can be done to deal effectively with such cases in hospitals; the matter rests entirely with the officers of their units.

Prognosis.—This, so far as it can be given, is based entirely on figures. There is no more to say than that so many patients became sufficiently well in hospital to be returned to duty, within the prescribed period of a month, and that so many others did not.

All patients admitted, and their disposal, are here set out in tabular form.

The competition between various centres, and rival methods of treatment for the return of patients to their units is a most vicious procedure in the treatment of functional disease, where there is no visible lesion, and symptoms are so largely subjective. With cases of

fractured femur a successful surgeon has cases of good union, absence of shortening, and general serviceability of limb to show, but cases of neurasthenia who are beginning to recover look very much like normal people, and can be sent to their units, when they count as "cures," although they may only add to the paper strength of a unit and readily relapse on exposure. Still, there is no other means of forming an opinion as to prognosis than that yielded by figures.

Of 1,300 cases 354 were either organic or had some intercurrent disease, or grave visceral symptoms, such as persistent incontinence or disordered action of heart, and since no female nurses were employed at this unit, and facilities for the comfort of seriously ill patients were few, the great majority of these (303) were transferred to other hospitals.

There remain 946 cases of uncomplicated functional nervous disease; of these 612 were "acute" cases of sudden onset, and 334 of gradual onset. Methods of discharge were the following:—

A. to duty with their units, generally with a recommendation for light duty for a period of a month.

B. to base duty, employment away from army areas.

C. to convalescent camp, for ultimate discharge to duty. This channel of discharge was only available towards the end of the period under consideration.

D. to base hospitals.

Discharge to	A	B	C	D
Acute cases, 612	307	104	103	98
Percentage	50 %	17 %	17 %	16 %
Gradually acquired, 334	68	140	44	82
Percentage	20 %	42 %	13 %	25 %
All cases of uncomplicated functional nervous diseases, 946	375	244	147	180
Percentage	40 %	26 %	15 %	19 %

That is, of all cases about 40 per cent. went direct to duty, about 40 per cent. to light duty or prolonged rest previous to duty, and about 20 per cent. to base hospitals.

Of the acute cases 50 per cent. went to duty, 34 per cent. to light duty, 16 per cent. to base hospital, and of the gradually acquired conditions 20 per cent. to duty, 55 per cent. to light duty, and 25 per cent. to base hospital.

There are, of course, right ways and wrong ways of treating these patients, but I look upon these results as being due chiefly to the natural recuperative power of healthy men given a reasonable chance.

I have records of forty men sent to duty who broke down within the next few months, but I have no doubt whatever, from what I have seen of relapsed cases treated in other centres, that there were many others. You cannot treat a man so well that you can guarantee him against relapse, but you can very easily send him up in an unfit condition. Nobody's judgment is perfect, and everybody will make mistakes in plenty, but, with the existing demand for men in the combatant units, it has been the duty of medical officers to risk error rather in the direction of sending up possibly unfit men than in that of losing possibly fit men to their units.

Treatment.—War-neurasthenia has been defined above as a condition of fatigue with loss of emotional and physical control. It thus contains physical and psychical factors, and the same principles of treatment are applicable to both.

The obvious treatment of fatigue and loss of physical control is rest and graduated exercise, and the same general idea is carried out psychically by removal from the causes of the condition, the maintenance of discipline and graduated mental occupation.

The general method employed with the cases was as follows: It was to be understood by the patients that they were not invalids in the rdinary sense, they were not ill, they had no wounds, no infection, nothing wrong with heart or lungs or other organs; they were merely strained and exhausted, and would soon recover. The graver cases were kept in bed for a few days—as a rule, not more than one—for a short complete rest, a dose of bromide of potassium was often given at night. This rest was not continued more than a day or two on account of the impression of invalidism given thereby, for many of the patients have "thrown down their cards," and are quite ready to accept the rôle of invalids. This is particularly the case with paretics who have lost the power to walk, and, naturally enough, think that they have lost it altogether, a sufficiently depressing thought. When such cases are got up, and made to walk and realize that they are not cripples, the improvement in their condition is often strikingly rapid; the same is true of a mute when he finds himself restored to human fellowship.

A sick parade was held daily at the medical officer's tent, which all patients attended on alternate days; this was the first step towards convalescence. The men were then employed on light fatigues about the tents, next attended physical drill under a trained instructor, next went out for route marches about the country, and finally moved to the convalescent section, which provided working parties for the heavier

fatigues required about the hospital. This was a fairly satisfactory general arrangement from the physical point of view, and worked pretty well; psychically it was incomplete. In this, as in the treatment of all other conditions, the prime necessity is the removal of the cause, and where people have broken down from exposure to shell-fire it is correct treatment to remove them to an area which is free from shell-fire and bombing raids. The setback which ensues on a hostile air-raid is very striking, the difficulty of maintaining discipline is extreme, and an excuse is provided for all who are inclined to exaggerate their symptoms. The Adjutant-General's branch, however, decided to keep these cases in centres in Army areas, which has added very greatly to the difficulties of their treatment. On the other hand, if the return of patients to their units is contemplated, it must be clearly understood throughout the hospitals that this is to be the case, and centres are not easily worked at bases on the coast, where the cliffs of England are easily to be seen, and from which patients are constantly being evacuated home. Neurasthenics, who have had their belly-full of fighting and have no desire to do any more, when they once find their condition has brought them to the base are likely to have continued subjective symptoms under these circumstances. The most suitable places for the treatment of cases likely to be fit to return to duty would be inland sites in France, altogether out of reach of bombing raids, and quite out of the way of artillery ranges, trench mortar schools and the like. Such not being available the best has to be made of what is to be had. One very common cause of psychical distress is domestic worry, such as the health of wives, the death of children, whom the fathers have sometimes never seen, the poverty of parents, the death in battle of brothers and intimate friends, and the occasional infidelities which are inevitable in long periods of marital separation. This of course opens up a very large question, that of leave, which has consequences far beyond the medical, but it has a very direct bearing both on the incidence and the recovery of cases of war-neurasthenia, and on the rare occasions when it is possible for a patient to obtain leave the opportunity should not be missed.

Discipline, however, should be very strictly maintained, and no kind of slovenliness permitted, but harshness is a thing altogether to be avoided. I have seen a large number of patients who were doing fairly well until they came under an officer who frightened them, and gave them the impression that he was bullying them, when they went to pieces. This is particularly the case with men suffering from defects in co-ordination, tremors and imperfect gait. At the same time, firm-

ness and even severity are essential with patients who will not make the necessary effort, and are ready with plausible excuses, and the distinction between severity and harshness is a very nice but a very real one. It can hardly be set down in words, but the guide is this: whatever gives you improvement, whether kindness or severity, is right; whatever puts your patients back and does not get them on is wrong, encouragement is always necessary. The matter of occupation is a difficulty; men ask for "anything to occupy my mind," but actually cannot maintain interest for any long period. The best results are given by getting men to work at their own skilled trades, carpentry, sign-writing, and gardening and the like, but it is difficult to get enough interest taken in ordinary "fatigues" for them to have much curative value. Concerts and cinema entertainments of an elaborate character are not of great value; patients frequently do not care for them, their headaches get worse and they come out in the middle. The best are those in which the patients take part, or which they arrange. Much might doubtless be done by personal interest in devising games, entertainments and occupations generally, but of this I have little experience from lack of the necessary gifts.

A matter constantly discussed in the psychical treatment of neurasthenia is suggestion; it is commonly said that the patients are suggestible in a high degree, and that hypnotism is of especial value. I do not employ hypnotism, and, having no experience of it, I have no criticism to offer upon it. In the treatment of the physical disorders of war-neurasthenia, which is the subject of this paper, it is chiefly of value, from what I am able to learn, in hysterical cases, but from the ease with which hysterical symptoms are treated by other methods I think it can rarely be necessary. I gather that in the inco-ordinate cases, which are the difficult ones, hypnotism is of little avail. But hypnotism apart, what is meant by the word "suggestion"? It appears to me to be used in medicine, perhaps unconsciously, in a sense quite foreign to that in which it is used in conversation. The word is used colloquially in such senses as the following: "I had suffered for a long time from abdominal pain, my doctor *suggested* an operation." "An apple falling from the tree *suggested* to Newton the theory of gravitation," "A scientific paper full of *suggestive* ideas." "I was continually missing money; he *suggested* that I was being robbed." All these uses of the word imply putting a new idea into your head, upon which you may on your own initiative take independent action. They imply that your mind was a blank on the subject before, not that you were seeking information on

which to take action; if the doctor suggested an operation, you had not thought of it before, but expected medicine to be sufficient treatment.

But the word "suggestion," as applied to the treatment of functional nervous symptoms, is used so as to convey an impression of the occult. Its practitioners are supposed to possess a mysterious power which is out of the reach of common men, an influence on others' lives to which few can pretend. Whether this is deliberately cultivated or not, I do not know, but it is the idea which exists in the public mind. Patients with functional symptoms come for treatment, and in some subtle fashion it is introduced into their minds that the power they thought they had lost is really present, and thereby the symptoms are relieved. It is also stated that neurasthenic patients are more suggestible than other people, and some curious examples of this are given. I have heard of a patient who came to hospital with a long list of symptoms entered on his field medical card; his medical officer read these, and, intending to chaff him a little, said: "By Jove, you seem to be in a pretty bad way," and was then called away. On his return he found the patient in the lowest depths of depression, his remarks having been taken quite literally. And this is an instance of suggestibility. It appears to me that it was merely expert confirmation of the patient's worst fears; had he had a sore on his tongue, and been told that he must undergo an immediate operation for excision, and been proportionately depressed, it would have been thought perfectly natural; the two cases seem to me exactly parallel.

Personally I do not think neurasthenics are more open to suggestion than anybody else, but they are in the main depressed, and their depression is increased if, when they come for information about their condition, that information is of a depressing character, and they are cheered up if the information is cheering, in which they behave in precisely the same way as other people. I propose, therefore, to omit suggestion as a point of great importance in the treatment of the physical symptoms of war-neurasthenia, regarding the term as a misnomer for the kind of psychical influence required. Apart from mere passage of time, and natural recuperative processes, these patients recover their lost faculties through their own efforts, and the necessary attitude of the medical officer is one of encouragement. This attitude is required throughout, but, perhaps, most conspicuously in the treatment of hysterical symptoms, paresis and anæsthesia; and encouragement is, in my opinion, far more effectively given by way of demonstration than by way of suggestion. The difference is this: "You think

you cannot walk, but I now put it into your head that you can," as compared with "You think you cannot walk, follow my instructions; observe you are walking." Exponents of hypnotism have told me that this is suggestion; I am unable to see it. In a case of anæsthesia the patient cannot feel ordinary stimuli, but he can feel stimuli sufficiently strong; when, therefore, I apply a powerful faradic current which he does feel, and after this ordinary stimuli are appreciated, I realize that the patient has had his capacity to feel demonstrated, but I fail to see that suggestion has anything to do with it. I am also unable to support the view sometimes expressed that the hysterical symptoms themselves are due to the suggestion of the doctor.

I have laboured this point somewhat because it seems to me important that those who have to treat such cases should not spend their energies on a misconception, when they have an efficient means of treatment to hand all the time: let them try demonstration.

The treatment applicable to several symptoms was stated when they were described, and are here recapitulated for the sake of completeness.

Pain is better relieved by physical appliances than by drugs. Heat, cold, fomentations and hot-water bottles, icebags and compresses, massage and blisters are all of value. For headache, the pillows should be packed at night, change of position should be made slowly, and in severe cases lumbar puncture may be considered, and headache due to spasm of accommodation may be relieved by atropine.

Anæsthesia of the skin and deep tissue may generally be left alone; if obtrusive, faradisation sufficiently strong to be felt should be employed.

Special senses, blindness and deafness. No cases of the former occurred in this series, but in a case reported to me from the ophthalmic department, vision was restored under examination with the ophthalmoscope, that is, by application of highly concentrated light to the retina.

Deafness often recovers spontaneously; loud unexpected sounds, such as a bell rung behind the head, and also the concentration of sound through the stethoscope often restores hearing; in deaf mutes the recovery of speech is of great assistance.

Defects of co-ordination, tremors and stammers. These are among the most difficult troubles to treat, where they are persistent and do not subside spontaneously.

For tremors, re-education, the performance of controlled movements under supervision, is of service, and perhaps the best results are obtained

when the services of a professional athletic trainer are available, who gives massage and systematic exercises. I have only seen this in practice on a few occasions but the results appeared to me to be good, and at any rate the principle is right. Faradization is sometimes of great value in genuine tremors, and checks assumed tremors very quickly.

Stammering. This often ceases spontaneously, sometimes quite abruptly, like the recovery of speech. But in many cases recovery is extremely slow, and persists at any rate for many months without improvement.

The treatment is re-education, and is often disappointing. The results vary with the amount of trouble taken by the medical officer and by the patient, and it is perhaps best expressed by saying that a patient who is intelligent enough to follow instructions, who will understand what to do when he is shown how to use his mouth for articulation, will benefit, but a stupid patient will not. The method adopted is as follows: the patient and the doctor sit opposite one another, their knees nearly touching. Breathing is first practised, deep breaths in and out, the patient taking time from the doctor. Instructions are then given as follows, "Fill your chest, a deep breath, now say 'A'." If there is hesitation, beat time by striking your hand on the patient's knee and say, "Say 'A' as I bring my hand down." Faradization by bringing an electrode on to the patient's hand in the same way will sometimes induce the necessary effort. If the patient repeats "A-A-A-A-," check him, and try to cut him short at the first syllable. Then take the other vowels, explaining the diphthongs if required, e.g., "I" = "AH-EE."

When these are going fairly well, take the consonants, giving instructions how they should be spoken.

"B." Take a deep breath, close your lips, burst them open and say "EE."

"C." Deep breath, draw back your lips, hiss through your teeth, say "EE."

"D." Tip of your tongue against the roof of your mouth just behind your teeth, pull it away and say "EE."

"F." Say "EH" and finish by putting your lower lip against your upper teeth.

"G." Tongue flat against the roof of your mouth, pull it away and say "EE."

"K." Back of your tongue against the roof of your mouth, pull it down, and say "A," and so on.

After this, the patient is to count up to ten, and after that he is asked to answer simple questions, such as name, number, regiment, service, civil employment and the like.

He may then pass on to repeat some familiar consecutive passage, such as the "Lord's Prayer."

For all this you require the patient's fullest attention, and must give your own. The treatment should be given in a separate room; if this is impracticable the end of a ward should be cleared of other patients and screened off. Patients are shy of exhibiting their disability, and you will not get them to do their best until they realize that you are entirely in earnest and will not allow them to be made ridiculous in any way.

Paresis.—You have here to demonstrate to the patient that he possesses the power which he believes himself to have lost.

The commonest form of paresis is that affecting the legs, with inability to walk; it is rarely so extreme that no movement at all is possible.

The patient should lie in bed, with the legs stripped. Examine the joints, muscles and reflexes to exclude organic disease. Hold your hand about 18 in. above his ankles and tell him to touch your hand with his foot: he will probably raise his foot a few inches, and if he fails to touch your hand, pick up the legs and move all the joints passively through their normal range, and tell the patient to repeat the movement; there will probably be some improvement, encourage this at once by praise. Lift your hand higher, and help his movement by lightly slapping the calf with your other hand. Stick to every movement till it is performed. In resistant cases great help is given by faradization: the symptoms are due to the fact that the patient cannot of his own unaided volition transmit the necessary impulse to his limbs. The cutaneous stimulus arouses him to make the necessary effort. Many patients are very frightened of the battery, and in these cases it should be applied at first quite weak and gradually increased, till it is strong enough to produce the required result.

Having demonstrated that the patient can move his legs naturally, the next thing is to get him on to his feet. Tell him to rise to a sitting position on the edge of his bed, helping him if required. He will probably be unable to get up on his feet, but will make some effort; help him as much as is necessary, but as little as possible. Once on his feet there is a general inclination to collapse and subside on to the floor, and here strong insistence is required; if he falls, you must pull him up at

once. He must be instructed to stand on a wide base, with the feet about 15 in. apart, and to stiffen the knees; in most cases he will gain his balance in a minute or two, he will stand unsteadily, but still he will stand. Point this out, and encourage him. Now stand facing him and hold his hands, assure him that you will not let him fall, and make him take a step towards you, insisting and encouraging all the time.

If progress is not rapid, stop and apply the battery again, and repeat. Demonstration, insistence and encouragement are required the whole time. When he begins to walk, walk backwards yourself in front of him to give him confidence, any attempt to subside on to the ground, or to totter backwards can generally be checked by sharp insistence. I remember no case that was not able at least to stand after a single session of treatment, and the great majority were able to walk back to their tents. It is important not to overtire these patients, many are put back by doing too much at first; it is essential to exercise them every day, each day doing a little more than previously. It may take weeks before there is complete restoration, and the earlier the case is taken in hand the better the prognosis.

Monoplegias are treated on similar lines.

Mutism.—This sometimes clears up spontaneously, and all degrees of difficulty are found in curing it; some patients have merely to be shown how to speak, and recover at once, others may take hours.

I have used the following plan of treatment.

Sit face to face with your patient, ask him to speak; he will indicate that he cannot do so but will show readiness to write; do not allow him to do this. Ask him to cough, the majority can do so quite easily; assure him that if he can cough he can speak, because it is using the same part of his throat. Then tell him to cough, and as he coughs say "A"—cough out "A." This is often successful at once, or in a few minutes. If there is delay or resistance, the battery is again very useful in stimulating to the necessary effort. I generally apply it to the upper part of the chest. If phonation presents great difficulty whispering is often practicable. Seize upon whatever the patient can do, and work up from that. As in all symptoms of the kind, insistence and encouragement are of the first importance.

When some phonation or whispering is possible, take the vowels and then the consonants as suggested under the treatment of stammering. It will often be found that in repeating any sequence like the vowels the voice rises higher and higher in the scale, but this subsides in a short time.

When whispering is correctly done, but there is difficulty in phonation, and it is not restored by the battery, it is often of service to use the laryngoscope. The presence of the mirror at the back of the throat often supplies the required stimulus; if necessary the laryngeal electrode may be used.

For visceral symptoms of severity there is very little to be done under the conditions prevailing in the neurological centres.

Cases of tachycardia were as a rule transferred to a special centre established for such patients, where they were treated for disordered action of the heart on principles of graduated exercise, with a large proportion of good results.

Respiratory symptoms require no special comment.

The alimentary symptoms, anorexia, nausea, vomiting, dyspepsia, constipation and diarrhœa, are to be treated on the usual lines by diet and the usual medicines. I have found none of any special value, and on the whole these patients are very insusceptible to drug treatment, the symptoms varying with the general condition.

The genito-urinary symptoms are equally resistant; a few cases of precipitancy and incontinence appeared to be benefited by bromide of potassium and tincture of belladonna, but by no means all. Seminal emissions seemed definitely better after the administration of iron and arsenic.

Several drugs have been tried for sleep. I have found no specific, but one or other of the following generally gives improvement: Potassium bromide, up to 30 gr.; potassium bromide, 15 gr.; chloral hydrate, 15 gr.; tr. opii, 15 ♏; paraldehyde, best given in half-drachm doses; trional, 10 gr.

But the essential treatment for the whole condition is initial rest, graduated exercise and occupation.

Hypothesis as to organic basis of functional nervous disease.—No one can be in contact with functional nervous disease for any long period without inquiring why the symptoms occur. What is the difference between a man's nervous system before and after he has developed neurasthenia, accepting the assumption that no visible change occurs? I was aware of one physiological fact which had some bearing on the question, namely, that in an animal under the influence of fear there is an increase of sugar in the circulating blood. This was explained on the assumption that in fear something is perceived in the environment for which a violent muscular effort

is required, either for the purposes of combat or escape, and that there is a physiological means of providing an additional supply of sugar, as fuel for the muscles, to enable this effort to be made. I was aware, further, that the increase was believed to be due to the activity of the adrenal glands, acting under the influence of the sympathetic system.

I wished therefore to find out how far the ductless glands might influence the condition of neurasthenia, but I had no facilities for research in this direction, and such published works as were accessible to me were of no assistance. Beyond finding sugar in the urine of a severe case of shock I had no data bearing on the subject.

On one occasion, however, the hospital was visited by Major Crile, of the U.S.M.O.R.C., the well-known authority on shock, with whom I was able to discuss the matter. Following up his suggestions, I have attempted to construct an hypothesis to explain the symptoms of neurasthenia. I do not wish to father these views on to Major Crile, as they are merely conjectures which have occurred to me as the result of his remarks. The thing, as far as I am concerned, is pure guess-work, unsupported by experiment, but would, perhaps, be worth experimental investigation.

The idea, at any rate, is as follows: Life depends on adaptation to environment, and in the higher animals the function of adaptation is discharged by the nervous system, consisting, as it does, of organs of sense, afferent nerves, centres and efferent nerves, which end in muscles. Adaptation consists in the reception of stimuli from the environment by the organs of sense, their interpretation at the centres, and the performance of appropriate movement by the muscles. Its simplest instance is the pure reflex, as, for instance, the contraction of the pupil, which is the adaptation of the eye to the environment of light; it is unconscious and involuntary, and cannot be prevented by the will. Closely allied to this, but depending on consciousness, is instinct, using that word to express impulses to action, which have to do with the preservation of life. A primitive instance of this is flinching from pain; if you accidentally touch a hot stove, you instinctively but consciously withdraw your hand; it is a conscious, instinctive impulse to action for the preservation of your person. Comparable to this, although more complex, is the instinct to escape from danger, as when a soldier sees bursting shells, recognizes these are dangerous to life, and feels the impulse to get to a place of safety. But we are not moved only by reflex and instinct; we are moved also by emotion, which I do

not here attempt to discuss, and by reason. Reason depends on previous experience and training, and hence on memory; it is an impulse to selected action, to secure an ultimate object, which obedience to instinctive action might imperil, and, consequently, for reasoned action, such actions must be controlled. The desire to escape from danger is inevitable, although of variable intensity in different individuals, but the muscular movements necessary to discharge such action may not be allowed to take place.

It is suggested that herein lies the physiological basis of neurasthenia. Major Crile's views, if I have understood them, are somewhat as follows: When anything appears in the environment which is recognizable as a source of danger, impulses are received and interpreted by the brain, but those impulses cannot pass from the sensory to the motor side of the brain, and so induce action to be taken, except in the presence of secretions from some of the ductless glands, of which the adrenal is probably one. On perception by the sensorium of the alarming object, impulses pass to the adrenal by way of the sympathetic, and induce it to secrete, and when this has occurred impulses can pass to the motor side of the brain, and appropriate action can be taken. Evidence of this action of these glands is the hyperglycæmia and occasional glycosuria which are found among these cases. Now a soldier under discipline has to suppress his instincts and act according to reason, the ultimate result to himself and his side of obeying his instinct to escape being much worse than if he kept his instinct suppressed. But that will not stop his ductless glands from working, and it is suggested that war-neurasthenia, the exhaustion, fatigue, loss of control, and visceral disturbances are due either to exhaustion of, or intoxication with, the products of the ductless glands, which have been under constant stimulus, but whose products have not been properly metabolized by the actions they were intended to facilitate.

I do not propose to consider the bearing of this on emotion, but tears, the excessive secretion of the lachrymal glands at distressing sights, and mouth watering, profuse secretion of the salivary glands in the anticipation of food, are instances of obvious glandular secretion on the perception of particular circumstances in the environment, and I suggest that the symptoms of war-neurasthenia may be referable to either over-stimulation or exhaustion of the ductless glands, especially the adrenal.

In support of this, Starling states that the injection of adrenalin arouses every activity that can be normally excited by the stimulation

of the sympathetic system, and the chief results of stimulating the sympathetic are the following: Vaso-constriction of the blood-vessels, dilatation of the pupil, increased secretion of sweat and saliva, acceleration of the heart, vaso-constriction of the intestines and kidney, vaso-dilatation of the same, inhibition of the intestines, contraction of the ileo-colic sphincter, and inhibition of the bladder.

Compare with this list the following symptoms noted in this paper, dilatation of the pupil, occasional sweating and salivation, tachycardia, the frequent occurrence both of constipation and diarrhœa, and the remarkable variations between polyuria and suppression. So far, therefore, as general clinical observations can support any such hypothesis, I think the experience gained from these cases goes to support the view that the symptoms of war-neurasthenia are due in part, at least, to prolonged over-stimulation of the ductless glands, chiefly the adrenal.

Conclusion.

No one can spend a year in charge of cases of war-neurasthenia without being convinced of the reality of this condition, of its severity and of its amenability to treatment. What the nature of the change produced in the ductless glands, if any, may be I have no idea, but I am convinced that it requires a long time for recovery, and I doubt if anyone who has once developed it will ever be fit for front-line soldiering within the time limit of any war. In cases primarily due to shock there is probably little change, and many men get quite fit for service. I do not think there is any means of testing this except by returning men to their units, when many are able to carry on, though they are not the men they were. After a second breakdown I think it is useless to send men up again, they are only adding to the paper strength of battalions and are quite unreliable in any stress, and probably do little but harm in showing a bad example. It is the duty of all medical officers to set sentiment aside and have nothing in mind except the fighting strength of the Army, but it must be the fighting strength. Record nursing, which claims to return to the line a greater percentage by two or three than the man at the next Army Centre, is a procedure in my opinion wholly to be deprecated, however much favour it may win with the administrative staff.

I would put in a very serious plea for a thoroughly sympathetic and intelligent grasp of the problem on the part of the higher medical authorities. It is not every doctor who knows how to treat functional

nervous disease; he requires training for it, just as he does for taking out teeth. The most influential man with whom the potentially neurasthenic soldier comes into contact is the battalion medical officer. Of this I am convinced, although I speak only from hearsay. Records of cases sent down as "shell-shock" or "neurasthenia" by different medical officers differ enormously; the few who know anything of nervous disease appear to have far the smallest numbers. A man who has the knowledge to recognize the man who merely dislikes it, and the character to send him back to the line, saves numberless soldiers and is of immense disciplinary value. Further, such a medical officer can pick out men who are beginning to get shaky and keep them under his own eye as orderlies or stretcher-bearers, and thus save many more, and his services are invaluable in dealing with a man returned to duty from hospital.

In the centres themselves it is of the first importance that a sufficient number of officers who know their business should be selected for the treatment, and that training to that end should be instituted. Preferably whole hospitals should be devoted to the work. Much more complete courses of treatment are required for patients who get past the centres. A man with a broken thigh receives, as he should, adequate supervision till he is well again, but the chances are much against his doing any further service. If these patients, with their tremors and stammers are allowed to drift, as they will unless it is somebody's business to see after them, a condition remains untreated which is at first curable, but rapidly becomes intractable and is then a serious disability for civil life.

My plea is for men whose disabilities are as real and as creditable as any wound, and for work which can only be undertaken by men properly educated, work which is tedious and exhausting, but which is of serious national importance, and which gives results.

HYPERGLYCÆMIA IN MENTAL DISORDERS.

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Introduction.

It has long been known that emotions, especially those of a depressive nature, have a bad influence on the course, if not on the origin, of saccharine diabetes.

In 1674, Thomas Willis wrote the following words, to which posterity has but little to add: "Interdum moeror & tristitia diuturna, item affectus convulsivi, aliaque spirituum animalium depressiones, ac mordationes, diathesin hanc morbidam parere aut fovere solent."

All who have since worked on diabetes agree on this point, that emotions, such as fear, trouble, anxiety and anger, often aggravate the illness (Senator, 1879; Frerichs, 1884; Naunyn, 1906; Lépine, 1909). Von Noorden (1917) also admits that emotions bearing a depressive character may cause an increase of glycosuria, though he does not believe in the nervous origin of real diabetes. For nervous glycosuria is based on hypersecretion of adrenalin, and diabetes mellitus is according to him a dyspancreatosis.

In the second place it is a remarkable fact that some persons, who are in a state of agitation, especially those of a neuropathic constitution, show a temporary excretion of sugar. They do not suffer from diabetes before, nor do they acquire it in the course of further observation. Of course, a good many of the patients, who show transitory glycosuria, appear to suffer from real diabetes in later years (von Noorden), but some of those cases can only be interpreted as examples of nervous glycosuria (cf. Goodhart, 1889; Flesch, 1891).

Thus we often observe glycosuria on the first day of admittance to hospital, although it cannot be found afterwards.

Ricci (1908) found transient glycosuria six times in fifteen cases, after the necessity of an operation was first communicated to the patients. Follin, Denis and Smillie (1914) found glycosuria after difficult examinations; the same was observed by Cannon and Fiske (Cannon, 1918, op. cit., p. 75) with excited spectators of a football match. Glycosuria in cases of grave psychical alterations are also well known; they are mere instances of the same kind of nervous glycosuria. I do not speak of cases of anatomical cerebral lesions, such as hæmorrhages, tumours, meningitis, &c., or even of dementia paralytica, but I refer to the merely functional psychoses. The fact that glycosuria is so often observed in melancholia was the starting point for my researches. I must refer to the first chapter of this paper, where this question is discussed in extenso.

I take it for granted that emotions of a depressive nature may cause glycosuria in man. Intentionally I do not speak of diabetes, but only of glycosuria. Now glycosuria may have two causes: either the amount of blood-sugar is too high (hyperglycæmia), or the kidneys allow the sugar to pass, though the percentage of sugar in the blood is normal (renal diabetes).

A priori it was probable that nervous glycosuria was caused by hyperglycæmia, for the kind of experimental glycosuria which is doubtless the consequence of an affection of the central nervous system—the famous "piqûre" of Claude Bernard (1854-1855)—depends on hyperglycæmia. The same is the case with the puncture of Aschner (1912).

Glycosuria after injection of diuretine, a caffeine derivate by which the nervous centres are stimulated, is also based on an increase of sugar in the blood (Rose, 1903; Nishi, 1909).

In both experiments ("piqûre" and injection of diuretine), glycosuria is brought about in the same way. That this glycosuria was totally dependent on the amount of glycogen in the liver was already demonstrated by Claude Bernard and affirmed by all authors who afterwards worked on this subject (see also Pollak, 1909). The stimulus in question, however, does not go directly to the liver, at least not exclusively, for glycosuria could not be procured, if the splanchnic nerves had been cut (Claude Bernard, 1877; Eckhardt, 1869), if both the suprarenal glands had been removed (A. Meyer, 1906, 1908; Kahn, 1911), or if those glands had been deprived of all possible innervation (Nishi, 1909; see also the experiments of Macleod and Pearce, 1911).

According to these various experiments, we have formed the following conception of the mechanism by which glycosuria is produced by "piqûre" and diuretine diabetes. From the central nervous system the stimulus is conducted by the splanchnics (principally the left; Nishi, 1909) to the suprarenal glands; this leads to hypersecretion of adrenalin, which is brought into circulation and reaches the liver, where more glycogen is mobilized. In consequence of this, more glycose is found in the peripheral blood (hyperglycæmia), and if the amount of sugar be high enough it can also be found in the urine.

That indeed splanchnic stimulation causes an increased secretion of the suprarenal glands was first demonstrated by Dreyer (1899) and confirmed by later observers, e.g., by Asher (1912, 1917); the secretion of adrenalin was proved in these experiments by its characteristic

influence on the blood-pressure. (See for an account of the literature on this subject Cannon, 1918, op. cit., pp. 41-43.)

Waterman and Smit (1908) found an increased percentage of adrenalin, not only after faradization of the suprarenal glands, but also after the "piqûre" of Claude Bernard, by testing the mydriatic power of the blood, drawn from the vena cava above the entrance of the adrenal veins on the pupil of the frog. These results could not be obtained by Kahn (1908) and Nishi (1909.)

That glycosuria, merely dependent on emotion, has quite the same mechanism as the "piqûre" and diuretine-diabetes was shown by Cannon, Shohl and Wright (1911). When a cat was extremely excited by a barking dog glycosuria appeared, whereas the urine was found without sugar before and after the experiment. By extirpation of the suprarenal glands this glycosuria could be prevented.

Cannon and De la Paz (1911) have also demonstrated by the same experiment that emotion causes an increase of adrenalin in the blood of cats.

The experiments by Elliot (1912) on cats by means of injections of β-tetrahydronaphthylamine hydrochloride form a valuable affirmation of Cannon's results. β-tetra gives a typical emotion of terror, and at the same time a decrease of adrenalin in the suprarenal glands, which could be prevented by cutting the splanchnics.

That emotional glycosuria in cats[1] is based on hyperglycæmia was proved by Scott (1917); emotional hyperglycæmia was also demonstrated in rabbits and dogs by Hirsch and Reinbach (1913, 1914), Bang (1913, No. 15) and Loewy and Rosenberg (1913, No. 121).

So we may take it for granted that both the "piqûre" diabetes and the so-called emotional glycosuria are based on hyperglycæmia, and that the latter is effected by a hypersecretion of adrenalin.[2]

It is impossible to mention all who have worked and experimented on the subject in question; I was, however, obliged to discuss the principal facts shortly, which led me to the investigations which I am now about to mention, facts which are moreover necessary in order to understand the significance of my results and the explanation I intend to give of them.

[1] This was first found by Böhm and Hoffmann (1878) in their experiments on "Fesselungs-diabetes," but it was not correctly interpreted until the publication by Cannon.

[2] That injection of adrenalin produces glycosuria was found by Blum in 1901, and confirmed by nearly all the authors who afterwards studied this subject.

Chapter I.—Researches on Blood-sugar.

Method.

It would lead me too far to discuss in extenso the technique of my researches. I used the method of Bang as described in his second publication on the subject (1916). The pressure of gas being very inconstant in these abnormal times, after an unsuccessful trial of four months, I used an electric boiling method, by which I obtained reliable results.

Instead of acetas uranylicus, I used nitras uranylicus, which is much better soluble.

As chloretum kalicum purissimum of Kahlbaum could not be had owing to the war, I used the same salt unpurified; I could not therefore use Bang's formula and was obliged to make my own standard, both for the self-reducing power of the solution of chloretum kalicum, as for the reducing power of solutions of 1, 1·5 and 2 per mille glycose.

In order to control my solutions I repeated these examinations every now and then and I always did so every time I used fresh solutions. During the first month of my researches I carried out every investigation twice, until I was sure of making no mistakes of any importance; afterwards I did so occasionally only.

At first I only examined the blood before the patients had broken their fast, but afterwards I also looked for alimentary hyperglycæmia, examining the patients before, and three-quarters, one and a half, two and a quarter, and three hours after they had breakfasted. In order not to surpass the limit of normal assimilation, I ordered a breakfast, consisting of 100 grm. of bread with butter and 200 c.c. of milk, which had also the advantage that psychical abnormal patients ate it with less reluctance than they would show by taking solutions of glycose, &c. There still remain some precautions to be taken, which I shall discuss afterwards.

Researches on Normal Persons.

Previously I examined ten normal men and ten normal women; the results can be seen in the following tables:—

These results tally exactly with the results of other authors, who worked with the method of Bang. According to Bang, the normal percentage of blood-sugar varies between 0·70 and 1·10 per mille. In his opinion, the sex does not make any difference worth mentioning, and this was the case with my results.

Elzas (1916), working by the same method, found the amount to vary from 0·80 per mille to 1·15 per mille (average 1 per mille). Ryser (quoted from von Noorden), 0·63 to 1·05 per mille (average 0·86 per mille). Von Noorden (1917), 0·70 to 1 per mille, usually 0·80 to 0·90 per mille.

Normal Men.

	Name	Age	Before breakfast Per mille	After breakfast ¾ hour Per mille	1½ hours Per mille	2½ hours Per mille	3 hours Per mille
1	d. B.	23	1·00	1·27	1·22	1·15	1·12
2	M.	38	1·05	1·27	1·19	1·21	1·05
3	H.	21	0·96	1·13	1·23	0·93	0·91
4	J.	23	1·07	0·93	0·96	1·03	0·98
5	v. d. H.	20	0·91	1·14	1·04	1·02	1·00
6	S.	20	1·09	1·05	1·09	1·08	1·10
7	L.	22	0·98	1·11	1·07	1·04	1·05
8	P. H.	26	1·03	1·13	1·07	1·16	1·20
9	V.	35	0·95	1·24	1·21	0·90	0·99
10	W.	54	0·87	1·07	1·13	1·01	0·86
	Average		0·99	1·13	1·12	1·05	1·03

Normal Women.

1	W.	25	0·94	0·99	0·97	0·90	0·93
2	S.	40	0·95	1·17	1·16	0·92	0·93
3	G.	22	0·95	0·96	0·98	1·03	0·87
4	S.	37	1·09	1·27	1·25	1·23	1·13
5	W.	20	1·04	1·14	1·12	1·07	1·09
6	H.	21	0·86	1·18	1·18	0·83	0·88
7	J. R.	22	1·0	1·13	0·94	0·96	0·91
8	v. L.	49	0·78	1·05	0·93	0·98	0·81
9	M.	22	1·0	1·28	1·11	1·08	1·19
10	de V.	14	1·06	1·24	1·30	1·25	1·28
	Average		0·97	1·15	1·19	1·02	1·00
	Average of both sexes		0·98	1·14	1·16	1·04	1·02

All these numbers refer to the amount of blood-sugar before breakfast.

After breakfast, Bang, and also von Noorden, found a small increase of the amount of blood-sugar. Elzas gave 150 grm. of bread and one cup of tea without milk or sugar, and found an increase respectively from 1·06 per mille before breakfast to 1·36 per mille one hour after breakfast; 1·08 to 1·30; 1·10 to 1·27; 0·80 to 1·28; 0·91 to 1·03; 1·07 to 1·38; 1·14 to 1·45; 1·15 to 1·42; 0·98 to 1·35; 1·09 to 1·38 per mille.

This increase is rather high; the amounts found by Elzas before breakfast were also higher than those of most other authors, as well as mine; moreover he gave more starch than I did.

There are some precautions which must not be neglected in these experiments.

(1) Of course the persons examined must not have any bodily diseases, which are apt to influence the amount of blood-sugar, such as cerebral lesions, infectious diseases, nephritis, great loss of blood diseases of the liver, &c.

(2) Also the temperature of the environment must be constant. Warm-blooded animals show an increase of the sugar percentage in the, cold (Bang); on the contrary, de Langen (1916, 1918) has observed a considerable increase of blood-sugar in animals and man in the tropics. Senator (1909) observed a constant increase of blood-sugar in rabbits, on which the "piqûre" of Sachs-Aronsohn was performed (puncture in the corpus striatum, where the temperature centre is localized); like de Langen, he could not observe glycosuria. I shall afterwards discuss this incongruity between hyperglycæmia and glycosuria (p. 280). As my examinations took place in the hospital, the temperature of the environment may be assumed to have been constant.

(3) The persons who are to be examined must not work, at least not heavily. For though the opinions of the various authors differ in this respect, it may still have some influence; probably light work gives an increase of blood-sugar, whereas exhaustive labour may reduce it (Bang; Bergsma, 1912). Most of my patients were bedridden, some were walking about without doing work of any importance. The normal persons, whom I examined, did no work at all.

(4) Finally the age is of importance for the blood-sugar. Also with normal persons the amount of sugar increases in advanced age (von Noorden, 1917). Laudenheimer (1898) found in psychoses above the age of 60 a ten times greater frequency of glycosuria than below the age of 60; Ehrenberg (1909) had the same results. As will appear from my tables, this factor has not influenced my results.

Researches on Psychoses.

I feel obliged to give a short clinical history of each patient I examined, as with our present knowledge of psychiatry I think it is not justifiable to mention the diagnosis only. Not only is the nomenclature widely different in the various countries and in the various publications even of fellow countrymen, but also the opinions about psychical diseases are still totally unsettled; quite different diseases have been described (and find their way into literature), e.g., by the name of psychasthenia, of dementia præcox, and also of melancholia. So, if in psychoses facts are stated, they must remain connected with the complex of symptoms in which they were observed; this is the only way finally to collect facts which belong together.

In my opinion, an exception is formed by epilepsy and dementia paralytica. In order to be short, I shall not give a clinical history in these cases. Of the sufferers from epileptic seizures, I have chosen those only who did not show any bodily abnormalities and who had already suffered for several years from well-observed general epileptic attacks, some of them being decidedly demented; they all represented types of the so-called genuine epilepsy.

For examples of dementia paralytica, I have also chosen undeniable types of this disease, all showing, besides the characteristic dementia, several of the well-known bodily changes (alterations of pupils, and tendon-reflexes, tremor, apoplectic and epileptic attacks, positive reaction of Wassermann in blood and cerebro-spinal fluid).

Having taken these precautions, I think it admissible to omit the history and to give only the results. I have principally examined cases of melancholia and of confusional insanity, and some cases of mania, whereas I have taken the other psychoses, dementia præcox, paralytic dementia and epilepsy, as a means of control only.

Blood-sugar in Dementia Præcox.

I examined ten sufferers from dementia præcox. It is a well-known fact that the opinions of the various authors differ widely with regard to this chapter of psychiatry. Even in the successive editions of Kraepelin's text-books different conceptions of dementia præcox are found, while Bleuler's (1911) definition of dementia præcox is still more comprehensive.

In my opinion, entirely different groups of psychoses are combined in this way. I have tried to avoid possible mistakes in classification by examining only types of the so-called hebephrenia, who all showed, as may be seen in the history, the well-known clinical symptoms of dementia præcox: the autism and loss of real interest in matters of daily life; the high degree of suggestibility on the one side and of negativism on the other, with all the abnormal movements and positions dependent on it; the incomprehensible associations in speaking and acting.

A second difficulty is caused by the fact that, in my opinion, it is quite impossible to make a diagnosis with absolute certainty from the complex of symptoms only, even if these symptoms are otherwise typical for dementia præcox.

In order to be able to pronounce a decisive opinion on diagnosis and prognosis, or to register symptoms as belonging to dementia præcox,

it is absolutely necessary that the disease be observed for a long time. For that reason I chose only patients who had already suffered from the typical symptoms for several years. No. 6 had been ill for the last half-year only, but then he had the typical symptoms and showed no signs of improvement at his departure.

No. 1.—F. P., female, aged 24. Two relatives at the second remove insane; further family history negative. Psychical alteration for three years, taciturn, doing no work, always staring; hebephrenic laugh; often cutting a queer face, not caring for her relations. In hospital the same symptoms. No bodily changes.

Blood-sugar.

August 20, 1918: Seemed anxious on admission. Pulse, 142; four hours after breakfast, 1·59 per mille.

August 21: Before breakfast much calmer. Pulse, 92; blood-sugar, 1·33 per mille.

August 22: Blood-sugar before breakfast, 1·20 per mille.

August 23: Blood-sugar before breakfast, 1·20 per mille.

August 24: Blood-sugar before breakfast, 1·07 per mille.

August 26: Blood-sugar before breakfast, 1·01 per mille.

October 9: Quite calm. Before breakfast, 0·89 per mille; three-quarters of an hour after breakfast, 1·07 per mille; one and a half hours after breakfast, 1 per mille; two and a quarter hours after breakfast, 0·98 per mille; three hours after breakfast, 0·91 per mille.

No. 2.—J. D., male, aged 38. Mother melancholic, further family history negative. Abnormal for seven years. Began with excitement, anxiety, aggressiveness spoke much about the Bible and Socialism, afterwards taciturn, laughing suddenly, staring with vacant eyes. Was in asylum for one and a half years. Did not recover.

In hospital reticent, many stereotyped actions, catalepsy, automatic movements. Slight dementia. No bodily changes.

Blood-sugar.

October 7, 1918: Quite calm. Before breakfast, 0·88 per mille; three-quarters of an hour after breakfast, 1·07 per mille; one and a half hours after breakfast, 1·14 per mille; two and a quarter hours after breakfast, 1·07 per mille; three hours after breakfast, 1·13 per mille.

No. 3.—H. P., male, aged 42. Father alcoholic, further family history negative. Patient has been abnormal from his 18th year, when he was in prison for some months.

Taciturn, said strange things, was always laughed at, earned little, occasionally stayed in bed for some days, often talked nonsense, several stereotyped movements.

In hospital reticent, apathetic, stereotyped movements, catalepsy. No bodily changes.

Blood-sugar.

February 15, 1919: Quite calm. Before breakfast, 0·77 per mille; three-quarters of an hour after breakfast, 1·22 per mille; one and a half hours after breakfast, 1·10 per mille; two and a quarter hours after breakfast, 0·97 per mille.

No. 4.—J. D., male, aged 21. One brother suffering from dementia præcox for several years.

Became more and more taciturn from his 10th year, stayed in bed, was quite indifferent to reproof, showing stereotyped actions, often staring.

In hospital behaving like a typical case of hebephrenia; reticent, stereotyped attitudes, hebephrenic laugh, &c. No bodily changes.

Blood-sugar.

February 15, 1919: Quite calm. Before breakfast, 0·81 per mille; three-quarters of an hour after breakfast, 1·22 per mille; one and a half hours after breakfast, 1·01 per mille; two and a quarter hours after breakfast, 0·92 per mille.

No. 5.—L. C. K., male, aged 28. Family history negative. Always a backward child (intellectually). Three years ago he became abnormal rather suddenly. Hallucinations, anxiety, persecutory delusions. Was treated in asylum and diagnosed dementia præcox, with symptoms of negativism, catalepsy, hallucinations and delusions. Never normal again, behaving strangely, childishly; stereotyped movements. Doing no regular work.

In hospital same symptoms. No bodily changes.

Blood-sugar.

November 16, 1918: Quite calm. Before breakfast, 0·99 per mille; three-quarters of an hour after breakfast, 1·13 per mille; one and a half hours after breakfast, 1·13 per mille; two and a quarter hours after breakfast, 1·02 per mille.

No. 6.—J. J. B., male, aged 22. Family history negative. Psychical alteration for about eight months. Taciturn, no interest in his surroundings, stereotypic, cataleptic, probably hallucinations, always staring with vacant eyes. Sometimes negativism. No bodily changes.

Blood-sugar.

September 24, 1918: Quite calm. Before breakfast, 0·99 per mille; three-quarters of an hour after breakfast, 1·17 per mille; one and a half hours after breakfast, 1·06 per mille; two and a quarter hours after breakfast, 0·94 per mille; three hours after breakfast, 1·16 per mille.

No. 7.—J. H., female, aged 21. Family history negative. Abnormal

for three years. Behaving strangely, stereotypically, without sense, laughing without reason, could not do the work she used to do.

In hospital the above-mentioned symptoms, typical schizophrenia. No bodily changes.

Blood-sugar.

October 8, 1918: Quite calm. Before breakfast, 0.76 per mille; three-quarters of an hour after breakfast, 1·16 per mille; one and a half hours after breakfast, 0·99 per mille; two and a quarter hours after breakfast, 0·85 per mille; three hours after breakfast, 0·76 per mille.

No. 8.—R. B., female, aged 52. Family history negative. Became abnormal on her twentieth year (after unhappy love affair ?). Since then never normal; occasionally excited, with hallucinations and delusions, always acting strangely, abruptly, living in complete bewilderment; had a collection of twenty-five pairs of shoes; on her first admission she wore seventeen skirts, eight under-vests, six chemises, three pairs of drawers, and three pairs of stockings; always talking, but complete nonsense only. The same psychical state on the second admission to hospital. Intellectual activity bad. No bodily changes.

Blood-sugar.

December 23, 1918: Continuously talking nonsense, no real excitement or emotion. Before breakfast, 1·13 per mille; three-quarters of an hour after breakfast, 1·18 per mille; one and a half hours after breakfast, 1·23 per mille; two and a quarter hours after breakfast, 1·07 per mille.

No. 9.—A. A. H., female, aged 28. Family history: both parents alcoholic. Psychical alteration since April, 1916. Began with anxiety, paranoid delusions, hallucinations; afterwards calmer, but behaving strangely, impulsively. After her first stay in hospital she neglected her household, often suffered from mutism; acted impulsively, quite indifferent to the death of her husband, became very erotic.

On second admission into hospital, all symptoms of dementia præcox. No bodily changes.

Blood-sugar.

September 24, 1918: Fairly calm, erotic. Before breakfast, 0·92 per mille; three-quarters of an hour after breakfast, 1·41 per mille; one and a half hours after breakfast, failure; two and a quarter hours after breakfast, 1·13 per mille; three hours after breakfast, 1·16 per mille.

No. 10.—M. S., female, aged 35. Family history negative. Psychical alteration for ten years. Taciturn, reticent; often staring; wandering away from home; negativistic; paranoid delusions, armed herself with stick; hallucinations. In hospital: reticent, occasionally negativistic, laughing hebephrenically, cataleptic. No bodily changes.

Blood-sugar.

December 18, 1918: Quite calm. Before breakfast, 1·16 per mille: three-quarters of an hour after breakfast, 1·85 per mille; one and a half hours after

breakfast, 1·08 per mille; two and a quarter hours after breakfast, 1·21 per mille.

December 21: Calm. Before breakfast, 1·17 per mille; three-quarters of an hour after breakfast, 1·54 per mille; one and a half hours after breakfast, 1·55 per mille; two and a quarter hours after breakfast, 1·22 per mille.

February 17, 1919: Calm. Before breakfast, 0·94 per mille; three-quarters of an hour after breakfast, 1·63 per mille; one and a half hours after breakfast, 1·15 per mille; two and a quarter hours after breakfast, 1·11 per mille.

If, in the case of No. 1, we take the observation at the time when she had become accustomed to the hospital, and if we take in the case of No. 10 the average of the three examinations, we may put down as *the average in these ten cases of dementia præcox* respectively:—

Before breakfast	0·93	per mille.
Three-quarters of an hour after breakfast	1·23	,,
One and a half hours after breakfast	1·10	,,
Two and a quarter hours after breakfast	1·10	,,

Two only of the ten cases showed a higher amount than 1·30 per mille after breakfast, of whom No. 9 was more or less excited. In the case of G. S. (No. 10) there was no obvious reason for the invariably increased amount of blood-sugar. She often blushed deeply, which fact, however, is not seldom seen in dementia præcox. I am inclined to think that emotions are responsible for the increase of blood-sugar in such cases, emotions which the patients are not willing or able to tell.

That emotions play a part, even with persons who afterwards seem to be the most indifferent types of hebephrenia, is shown by the examination of Case No. 1. This girl came into the hospital in a distinctly emotional state of mind, having a pulse of 142 and an amount of blood-sugar (four hours after breakfast) of 1·59 per mille. This percentage showed a decrease on the successive mornings: 1·33 per mille, 1·20 per mille, 1·20 per mille, 1·07 per mille, 1·01 per mille. The patient had become accustomed to hospital life and was quite calm.

The only way to solve the difficult problem of the psychology of dementia præcox is by noting the involuntary bodily changes which accompany psychical processes; for all reactions, noted during psychological examination of the patient, are only fit to demonstrate the abnormalities of the sufferer, not to get to the bottom of things. The examination of blood-sugar may also help us in this way. But, in my opinion, many examinations of dementia præcox patients are necessary

in many different conditions of mood, and also a careful comparison with other bodily reactions, before we are in a position to draw any conclusions.

These results correspond to those of Schultze and Knauer (1909), who found absence of glycosuria in the apathetic form of hebephrenia, whereas glycosuria was often demonstrated in cases of catatonic excitement, especially after anxious hallucinations. Travaglino (1911) described a catatonic patient suffering from glycosuria, which came and went parallel with depression, anxiety and hallucinations; he did not observe glycosuria in the hebephrenic form. Ehrenberg (1909) could not find any alimentary glycosuria in his nine cases of dementia præcox, neither could Graziani (1908) nor Lugiato (1907), whereas Trepsat (1905) observed it in eighteen out of his nineteen cases (five acute cases and fourteen chronic ones) after a dose of 150 gr. of glycose. [See, for literature, Allers (1913.)]

Heidema (1918) is the only one who examined the percentage of blood-sugar before breakfast in dementia præcox more carefully.

In ten of his thirteen cases he found a more or less marked hyperglycæmia, but nearly all these thirteen patients had only suffered from the illness for a few months or half a year, and judging from the author's notes the diagnosis was often not certain.

The possibility that some of Heidema's cases were not really sufferers from dementia præcox[1] and the fact that, if the diagnosis was correct, they certainly were examined in a more acute stage of their illness than my patients, may explain the difference between Heidema's results and my own sufficiently.

Blood-sugar in Dementia Paralytica.

According to the text-books of Kraepelin (1910) and of Church and Peterson (1914), intermittent glycosuria is a symptom which is occasionally met with in paralytic dementia. I do not believe that we can go any farther. Bond (1895) noticed glycosuria in 10 per cent. of the cases, Siegmund (1895) in 27·8 per cent., Strauss (1900) in 9 per cent., and Schultze and Knauer in 67 per cent. Especially the exalted form showed glycosuria, not that of simple dementia. The percentages

[1] Perhaps some cases belonged to maniacal-depressive insanity or to that large and still undefined group of amentia and amentia-like diseases, in both of which I found a high amount of blood-sugar; Raimann (1902) also found a very low limit of the assimilation power in amentia, even lower than in melancholia.

given by the latter authors are doubtlessly too high.[1] Raimann (1902) did not find glycosuria in paralytic dementia, nor did Heidema (1918). Raimann did not find a marked influence on the assimilation power, though the depressive forms showed a lower number than the rest. Strauss observed alimentary glycosuria in 9 per cent. of the cases, Arndt (1897) in 10 per cent. Finally we have the researches of Heidema, who found a small increase of blood-sugar before breakfast.

My own results are the following :—

	Name	Age	Sex	Before breakfast	¾ hour after breakfast	1½ hours after breakfast	2½ hours after breakfast	3 hours after breakfast	Notes on psychical state
				Per mille	Per mille	Per mille	Per mille	Per mille	
1	B.	58	M.	0·89	1·54	1·30	1·01	1·09	Quite calm in bed ; extreme dementia
2	D.	35	M.	1·08	1·35	0·95	0·84	0·86	Merely dementia ; quite calm in bed
3	de H.	44	M.	1·07	1·45	1·24	1·41	1·20	Terminal stage ; lay sucking in bed
4	H. B.	44	M.	0·95	1·32	1·32	1·41	1·14	In merry mood ; delusions of riches ; walking about
5	de B.	41	M.	1·02	1·05	1·09	1·01	—	Calm, merely dementia ; walking about
6	N.	51	M.	1·01	1·27	1·24	1·05	—	Merely dementia ; calm
				1·36	1·36	1·63	1·35	—	During paralytic attacks
7	S.	38	M.	0·87	1·43	1·22	1·03	—	Severe attack of rage directly after breakfast ; afterwards calm
				0·83	1·16	1·05	—	—	Walking, calm, merely dementia
8	T.	46	F.	1·09	1·31	1·22	0·84	—	Calm in bed, merely dementia
9	K.	47	F.	1·09	1·55	1·45	1·19	—	Calm in bed, many absurd delusions of power and riches

If we exclude the results of Nos. 6 and 7, obtained in paralytic attacks and in excitement, we get the average :—

1·01 .. 1·31 .. 1·21 .. 1·09 per mille

the average of the *highest amounts* being 1·33 per mille.

[1] Both Siegmund and Schultze and Knauer examined the urine of every patient several times, but not equally often ; moreover, in different diseases, they did not examine the urine an equal number of times ; in this way many more sufferers from transitory glycosuria might have been detected in one illness than in another. Schultze and Knauer made another mistake in establishing their percentage by taking the quotient of the number of urine examinations and of the positive results. We not only cannot find how many sufferers from transitory glycosuria there are in the diseases in question in this way, but the percentage is also dependent on the number of examinations, and especially on whether the sufferers from glycosuria were examined more or less often than the non-sufferers.

The amount per mille is doubtless higher than that of normal persons, six of the nine patients having, without excitement or apoplexy, an amount above 1·30 per mille after breakfast.

The results do not disagree with the conclusions arrived at after studying the literature; the amount of blood-sugar usually does not reach the height where glycosuria becomes frequent,[1] but has a tendency to be above the normal.

As paralytic dementia is a disease with typical anatomical changes (to which, I think, the increase of blood-sugar must be attributed), we cannot use it for the question which for the moment arrests our attention.

Blood-sugar in Epilepsy.

I examined eight sufferers from genuine epilepsy. The results are the following:—

	Name	Age	Sex	Before breakfast	½ hour after breakfast	1½ hours after breakfast	2½ hours after breakfast	3 hours after breakfast	Notes on psychical state
				Per mille	Per mille	Per mille	Per mille	Per mille	
1	S. D.	59	F.	1·01	1·58	1·31	1·09	1·13	Very irritable, had a severe attack of excitement after breakfast; dementia
2	J. N.	24	M.	0·87	1·23	1·11	0·90	0·82	Quite calm; dementia
3	R. T.	15	M.	0·84	1·08	0·83	0·89	—	Quite calm; dementia
4	W. D.	33	F.	1·00	1·03	1·11	1·16	1·06	Quite calm; slight degree of dementia
5	M. W.	41	F.	1·02	1·36	1·05	0·95	0·97	Quite calm; dementia
6	K.	43	F.	0·90	1·15	1·12	1·15	0·93	Quite calm; no obvious dementia
7	R. I.	14	M.	0·78	1·09	1·00	0·84	0·86	Quite calm; slight degree of dementia
8	K. A.	27	F.	0·88	1·45	1·35	1·01	—	Dementia; very nervous; several little attacks during examination

If we exclude No. 1 (who was examined in an emotional state of mind) and No. 8 (who was very nervous, had several epileptic attacks during examination), we find for the remaining three men and three women rather subnormal amounts, the average of Nos. 2 to 7 being:—

0·90 .. 1·16 .. 1·04 .. 0·98 .. 0·93 per mille

and the average of the *highest amount* being 1·18 per mille.

[1] Siegmund also describes a case in which sugar appeared in the urine at the beginning of exaltation, and another case where it could be observed directly after a paralytic attack.

Only No. 5 showed an amount per mille above 1·30 (three-quarters of an hour after breakfast) for which no reason could be found. As far as these few observations enable us to draw conclusions we may say that genuine epilepsy itself does not increase the amount of blood-sugar, the momentary state during an attack perhaps excepted, and, of course, if not complicated by an intervening emotion.

This corresponds with the results of former observers as regards glycosuria or alimentary glycosuria.

Bond (1895) did not find glycosuria in epilepsy, neither did Strauss, Schultze and Knauer, van Oordt (1898), or Geelvink (quoted from Strauss).

According to Allers (1912), glycosuria is very rare in epilepsy; only Goolden (quoted from Allers) could observe it during epileptoid attacks, and Florence and Clément (1909) found a decrease of the assimilation power in epilepsy. Siegmund, who seldom found it with epileptic dementia, observed it after several attacks. On the contrary, Strauss and Schultze and Knauer could not even find glycosuria during attacks, and Raimann found in these circumstances a rather high assimilation power. Heidema found in four patients suffering from genuine epilepsy an average of 1·185 per mille, which is rather high in comparison with my results before breakfast. He attributes it to the fact that blood-sugar and glycosuria need not at all be parallel, which is true, of course, as I shall afterwards discuss. However, as we have no examination on blood-sugar in epilepsy, I was obliged to refer to those on spontaneous and on alimentary glycosuria, as being most germane to my researches.

Blood-sugar in Melancholia.

Of all the mental disorders, it is in melancholia that we have the most and best information about carbohydrate metabolism. This illness has been the starting-point for my researches. All nerve specialists know those cases where glycosuria appears at the same time with depression and disappears with psychical improvement; we find it mentioned in the larger text-books (e.g., of Kraepelin, Ziehen, or Aschaffenburg). A detailed discussion of the subject can be found in the papers of Allers (1914), of Schultze and Knauer (1911), of Mita (1912), and of Heidema (1918); so I think it superfluous to give a complete survey of the extensive literature.

Typical cases of glycosuria, dependent on melancholia, have been described, e.g., by Goodhart (1889), Bond (1895), Toy (1895), Schultze and Knauer (1911). The question of glycosuria in melancholia was

examined systematically by Schultze and Knauer, that of alimentary glycosuria by Raimann. The first mentioned authors found glycosuria in 67 per cent. of their cases. I refer to p. 227 for my criticism on their method of ascertaining the percentages. According to their researches, however, we may take it for granted that glycosuria is very frequent, even if we do not accept exactly the numbers given by them. For if we consider all those cases as positive in which the authors observed glycosuria at any time, we get a percentage which is much higher than 67 per cent. Especially in anxiety, glycosuria is seldom absent, in accordance with its frequency in emotional states of other diseases. I shall afterwards return to this question.

Raimann found a marked decrease of the assimilation power in melancholia, namely 1·9, the normal average being 3·5.

I have examined the following nineteen cases of melancholia; several of these patients were sufferers from real periodic melancholia; others had their first attack but in all of them there was genuine depression, with concomitant motor inhibition, anxiety, depressive delusions; in none was the depressive mood secondary, dependent on other deeper psychical or bodily causes, nor were there bodily changes or dementia in any of them.

No. 1.—P. de V., male, aged 65. Several members of his family suffered from maniacal-depressive insanity. Patient had had two attacks of melancholia before: depression, motor inhibition, delusion of having sinned; delusions often very absurd. No bodily changes, blood-pressure 130 mm. Hg (Riva-Rocci); examination showed poor intellectual powers, but patient had little school-learning and was very refractory during examination. Heavy drinker in former years.

Blood-sugar.

September 17, 1918: Calm, not agitated. Before breakfast, 1·09 per mille; three-quarters of an hour after breakfast, 1·15 per mille; one and a half hours after breakfast, 1·16 per mille; two and a quarter hours after breakfast, 1·30 per mille; three hours after breakfast, 1·17 per mille.

September 18: Same psychical state. Before breakfast, 1·07 per mille; three-quarters of an hour after breakfast, 1·17 per mille; one and a half hours after breakfast, 1·21 per mille; two and a quarter hours after breakfast, 1·18 per mille; three hours after breakfast, 1·28 per mille.

December 16: Same psychical state. Before breakfast, 1·20 per mille; three-quarters of an hour after breakfast, 1·36 per mille; one and a half hours after breakfast, 1·30 per mille; two and a quarter hours after breakfast, 0·98 per mille.

No. 2.—J. R., male, aged 66. Insane for the first time, became melancholic after the illness and death of his son; depression, delusion of having

sinned: high degree of anxiety; speaking and acting slowly; no dementia; no bodily changes.

Blood-sugar.

November 28, 1918: Very anxious. Before breakfast, 1·13 per mille; three-quarters of an hour after breakfast, 1·07 per mille; one and a half hours after breakfast, 1·20 per mille; two and a quarter hours after breakfast, 1·03 per mille.

December 3: Very anxious. Before breakfast, failure; three-quarters of an hour after breakfast, 1·83 per mille; one and a half hours after breakfast, 1·22 per mille; two and a quarter hours after breakfast, 1·02 per mille.

December 14: Very anxious. Before breakfast, 1·37 per mille; three-quarters of an hour after breakfast, 1·61 per mille; one and a half hours after breakfast, 1·17 per mille; two and a quarter hours after breakfast, 1·26 per mille.

No. 3.—H. J. P., female, aged 43. Twin brother committed suicide; father feeble-minded. Patient had already once suffered from melancholia. In 1918 she had several melancholic attacks of short duration; depression; motor inhibition; delusion of poverty; no anxiety; no dementia; no bodily changes.

Blood-sugar.

September 19, 1918: Calm, not anxious. Before breakfast, 1·10 per mille; three-quarters of an hour after breakfast, 1·42 per mille; one and a half hours after breakfast, 1·14 per mille; two and a quarter hours after breakfast, failure; three hours after breakfast, 0·91 per mille.

No. 4.—V. W., female, aged 60. Nervous woman, especially of late years. For the last months: Depression, motor inhibition, agitation, weeping all day; was never melancholic before; no dementia; no bodily changes; blood pressure, 130 mm. Hg (Riva-Rocci).

Blood-sugar.

September 19, 1918: Calm, no anxiety. Before breakfast, 0·93 per mille; three-quarters of an hour after breakfast, 1·41 per mille; one and a half hours after breakfast, failure; two and a quarter hours after breakfast, 1·22 per mille; three hours after breakfast, 0·96 per mille.

No. 5.—G. D. H., female, aged 23. First attack of melancholia; first child born three months before illness broke out. Depression, marked anxiety, delusion of having sinned, motor inhibition, sometimes stupor, attempt at suicide; intellectual examination impossible; no obvious dementia; no symptoms of dementia præcox. Scratching and plucking at everything, afterwards obstinate masturbation. No bodily changes.

Left hospital unrecovered.

Blood-sugar.

September 5, 1918: Anxious. Before breakfast, 1·07 per mille; three-quarters of an hour after breakfast, 1·69 per mille; one and a half hours after breakfast, 1·30 per mille; two and a quarter hours after breakfast, 1·09 per mille; three hours after breakfast, 1 per mille.

September 17: Less anxious, but still depressed. Before breakfast, 1·02 per mille; three-quarters of an hour after breakfast, 1·41 per mille; one and a half hours after breakfast, 1·45 per mille; two and a quarter hours after breakfast, 1·09 per mille; three hours after breakfast, 0·96 per mille.

No. 6.—B. F. de L., female, aged 41. First depressive period, but character was always melancholic. Became depressed after death of her mother. Depression, anxiety, delusion of having maltreated her mother; attempt at suicide; no dementia; no bodily changes.

Blood-sugar.

November 27, 1918. Depression, anxiety (slight degree). Before breakfast, 1·34 per mille: three-quarters of an hour after breakfast, 1·96 per mille; one and a half hours after breakfast, 1·75 per mille; two and a quarter hours after breakfast, 1·33 per mille.

December 7: Same psychical state. Before breakfast, 1·19 per mille; three-quarters of an hour after breakfast, 1·82 per mille; one and a half hours after breakfast, 1·53 per mille; two and a quarter hours after breakfast, 1·08 per mille.

February 4, 1919: Much better, but still very taciturn. Before breakfast, 1·18 per mille; three-quarters of an hour after breakfast, 1·66 per mille; one and a half hours after breakfast, 1·33 per mille; two and a quarter hours after breakfast, 1·39 per mille.

Left hospital not quite recovered.

No. 7.—M. M., female, aged 48. Melancholic depression four years ago. This time melancholia broke out after insignificant operation; depression; delusion of poverty, of having sinned; agitation; no dementia; no bodily changes.

Blood-sugar.

October 9, 1918: Agitated, bewailing her misfortunes. Before breakfast, 1·28 per mille; three-quarters of an hour after breakfast, 1·42 per mille; one and a half hours after breakfast, 1·32 per mille; two and a quarter hours after breakfast, 1·36 per mille; three hours after breakfast, 1·40 per mille.

December 6: Same psychical state. Before breakfast, 1·20 per mille; three-quarters of an hour after breakfast, 1·35 per mille; one and a half hours after breakfast, 1·43 per mille; two and a quarter hours after breakfast, 1·43 per mille.

Left hospital unrecovered.

No. 8.—D. L., male, aged 45. Mother committed suicide; several cases of insanity in family history. Patient suffered from melancholia twice before. This period: Depression, anxiety, depressive delusions, especially of a hypochondriac nature; some psychasthenic symptoms; no dementia; no bodily changes. Left hospital unrecovered; completely recovered in asylum.

Blood-sugar.

November 27, 1918: For psychical symptoms *vide supra*, anxious. Before breakfast, 1·19 per mille; three-quarters of an hour after breakfast, 1·62 per mille; one and a half hours after breakfast, 1·54 per mille; two and a quarter hours after breakfast, 1·44 per mille.

December 6: Same psychical state, anxiety. Before breakfast, 1·11 per mille; three-quarters of an hour after breakfast, 1·90 per mille; one and a half hours after breakfast, 1·39 per mille; two and a quarter hours after breakfast, 1·05 per mille.

No. 9.—A. v. d. W.—female, aged 38. First depressive period; slight depression; motor inhibition; no anxiety; no delusions; no suicidal tendencies; no dementia; no bodily changes; left hospital unrecovered.

Blood-sugar.

November 13, 1918: Calm, slight motor inhibition. Before breakfast, 0·92 per mille; three-quarters of an hour after breakfast, 1·31 per mille; one and a half hours after breakfast, 1·03 per mille; two and a quarter hours after breakfast, 0·91 per mille.

December 5: In the same condition. Before breakfast, 1·02 per mille; three-quarters of an hour after breakfast, 1·18 per mille; one and a half hours after breakfast, 1·34 per mille; two and a quarter hours after breakfast, 0·94 per mille.

No. 10.—A. M. J. M. H., female, aged 41. Melancholic for the first time; depression; depressive delusions; anxiety; no dementia; anæmia (had always looked pale, no change of the blood); no other bodily changes. Left hospital unrecovered.

Blood-sugar.

September 21, 1918: Depression; motor inhibition; no anxiety. Before breakfast, 1·04 per mille; three-quarters of an hour after breakfast, 1·33 per mille; one and a half hours after breakfast, 1·36 per mille; two and a quarter hours after breakfast, 1·28 per mille; three hours after breakfast, 1·23 per mille.

December 5: Same psychical state. Before breakfast, 1·07 per mille; three-quarters of an hour after breakfast, 1·53 per mille; one and a half hours after breakfast, 1·36 per mille; two and a quarter hours after breakfast, 1·13 per mille.

No. 11.—C. K. B., female, aged 50. Sister of our patient committed suicide. Patient suffered from melancholia once before. This time: depression,

agitation, anxiety, depressive delusions of all kinds, two attempts at suicide; no dementia; no bodily changes.

Blood-sugar.

November 13, 1918: Very anxious. Before breakfast, 1·35 per mille; three-quarters of an hour after breakfast, 1·62 per mille; one and a half hours after breakfast, failure; two and a quarter hours after breakfast, 1·24 per mille.

December 5: Same psychical state; anxious. Before breakfast, 1·27 per mille; three-quarters of an hour after breakfast, 1·70 per mille; one and a half hours after breakfast, 1·33 per mille; two and a quarter hours after breakfast, 1·27 per mille.

February 6, 1919: Stuporous; looking very anxious. Before breakfast. 1·20 per mille; three-quarters of an hour after breakfast, 2·04 per mille; one and a half hours after breakfast, 1·65 per mille; two and a quarter hours after breakfast, 1·10 per mille.

March 20: Recovered, about to leave. Before breakfast, 0·79 per mille; three-quarters of an hour after breakfast, 1·18 per mille; one and a half hours after breakfast, 0·98 per mille; two and a quarter hours after breakfast, 1·01 per mille.

No. 12.—J. M., female, aged 34. First attack of melancholia; depression; motor inhibition; no delusions; no anxiety; no dementia; no bodily changes.

Blood-sugar.

September 21, 1918: Slight depression and motor inhibition; no anxiety. Before breakfast, 0·99 per mille; three-quarters of an hour after breakfast, 1·33 per mille; one and a half hours after breakfast, 1·25 per mille; two and a quarter hours after breakfast, 0·98 per mille; three hours after breakfast, 1 per mille.

December 4: Depression; motor inhibition; weeping during examination. Before breakfast, 1·03 per mille; three-quarters of an hour after breakfast, 1·51 per mille; one and a half hours after breakfast, 1·19 per mille; two and a quarter hours after breakfast, 1·03 per mille.

No. 13.—E. de H., male, aged 44. Patient suffered from melancholia twice before. This time he shows: depression; motor inhibition; sometimes marked anxiety. Slight degree of tuberculosis of the lungs; temperature not raised; no anæmia; no cachexia; no other bodily changes. No dementia; melancholic, indecisive character.

Blood-sugar.

October 14, 1918: Depression; motor inhibition; no anxiety. Before breakfast, 1·13 per mille; three-quarters of an hour after breakfast, 1·61 per mille; one and a half hours after breakfast, 1·07 per mille; two and a quarter hours after breakfast, 0.89 per mille; three hours after breakfast, 0·84 per mille.

November 21: Depression; negativism; refusal of food. Before breakfast, 0·96 per mille; three-quarters of an hour after breakfast (only 400 c.cm. of milk), 1·51 per mille; one and a half hours after breakfast, 1·35 per mille; two and a quarter hours after breakfast, 1·06 per mille. Left hospital unrecovered.

No. 14.—F. K., male, aged 68. Had an attack of melancholia fourteen years ago: the outbreak of the present attack was preceded by a short period of excitement. Now: depression; motor inhibition, at the same time anxious; delusion of depressive nature, especially hypochondriacal delusions; no dementia; no bodily changes. Father of patient committed suicide.

Blood-sugar.

November 1, 1918: Psychological state: see above. Before breakfast, 1·13 per mille; three-quarters of an hour after breakfast, 1·57 per mille; one and a half hours after breakfast, 1·52 per mille; two and a quarter hours after breakfast, 1·39 per mille; three hours after breakfast, 1·24 per mille.

November 20: Depression; motor inhibition; not anxious; getting much better. Before breakfast, 1·03 per mille; three-quarters of an hour after breakfast, 1·17 per mille; one and a half hours after breakfast, 1·26 per mille; two and a quarter hours after breakfast, 1·22 per mille. Left hospital nearly recovered.

No. 15.—J. P. L.-S., female, aged 51. Melancholia (for the first time) some weeks after recovery from pneumonia. Depression; depressive delusions; motor inhibition; stuporous on admission into hospital; occasionally anxious; no dementia; no bodily changes.

Blood-sugar.

October 13, 1918: Excited; depressed. Before breakfast: 1·23 per mille; three-quarters of an hour after breakfast, 1·67 per mille; one and a half hours after breakfast, 1·60 per mille; two and a quarter hours after breakfast, 1·57 per mille.

November 21: Depression; motor inhibition; slightly anxious. Before breakfast, 1·45 per mille; three-quarters of an hour after breakfast, 1·82 per mille; one and a half hours after breakfast, 1·78 per mille; two and a quarter hours after breakfast, 1·50 per mille.

December 24: Patient has become maniacal; exaltation; accelerated flow of thoughts; still some depressive delusions. Before breakfast, 1·06 per mille; three-quarters of an hour after breakfast, 1·12 per mille; one and a half hours after breakfast, 1·09 per mille; two and a quarter hours after breakfast, 1·14 per mille.

January 6, 1919: Again depressed; depressive delusions; no motor inhibition. Before breakfast, 1·16 per mille; three-quarters of an hour after breakfast, 1·33 per mille; one and a half hours after breakfast, 1·43 per mille; two and a quarter hours after breakfast, 1·42 per mille.

No. 16.—L. S. B., female, aged 61. Sister committed suicide. Patient had one depressive period previously; this time: depression; anxiety; delusions of depressive nature; attempt at suicide; no dementia; no bodily changes. Left hospital not quite recovered.

Blood-sugar.

September 26, 1918: Anxious. Before breakfast, 0·78 per mille; three-quarters of an hour after breakfast, 1·52 per mille; one and a half hours after breakfast, 1·67 per mille; two and a quarter hours after breakfast, 1·53 per mille; three hours after breakfast, 1·21 per mille.

October 22: Day of departure, very nervous. Before breakfast, 1·28 per mille; three-quarters of an hour after breakfast, 1·73 per mille; one and a half hours after breakfast, 1·58 per mille; two and a quarter hours after breakfast, 1·09 per mille; three hours after breakfast, failure.

No. 17.—M. D. O., female, aged 47. Suffered from depressive insanity once before. This time: depression; very marked anxiety; delusions of depressed nature, especially of having sinned; attempts at suicide; refusal of food; no dementia; no bodily changes. Unrecovered to asylum.

Blood-sugar.

August 16, 1918. Before breakfast, anxious, 0·90 per mille.

August 17: Before breakfast, anxious, 0·88 per mille.

August 19: Before breakfast, anxious, 0·88 per mille.

August 20: Before breakfast, anxious, 1·20 per mille.

August 24: Before breakfast, anxious, 1·24 per mille; three-quarters of an hour after breakfast, 1·77 per mille; one and a half hours after breakfast, 1·33 per mille; two and a quarter hours after breakfast, 1·02 per mille; three hours after breakfast, 1·14 per mille.

August 26: Anxious. Before breakfast, 1·04 per mille; three-quarters of an hour after breakfast, 1·49 per mille; one and a half hours after breakfast, 1·43 per mille; two and a quarter hours after breakfast, 1·18 per mille.

October 23: Same psychical state; marked anxiety. Before breakfast, 0·93 per mille; three-quarters of an hour after breakfast, 1·46 per mille; one and a half hours after breakfast, 1·75 per mille.

Further examination impossible on account of anxiety. Except for the last time, no muscular exertion. Left hospital unrecovered.

No. 18.—L. M. v. W., female, aged 38. Father died from diabetes. Patient bore emotional character; insane for the first time; depression; marked anxiety; delusions of depressive nature; suicidal tendency; no dementia; no bodily changes. Unrecovered to asylum.

Blood-sugar.

August 16: Before breakfast, anxious, 1·07 per mille.

August 19: Before breakfast, anxious, 1·04 per mille.

August 20: Before breakfast, anxious, 0·87 per mille.

August 21: Before breakfast, anxious; much excited after having seen her child, 1·30 per mille.

August 22: Anxious. Before breakfast, 1·10 per mille; three-quarters of an hour after breakfast, 1·88 per mille; one and a half hours after breakfast, 1·89 per mille. (The patient had, however, taken 200 c.cm. of milk one hour after breakfast); two and a quarter hours after breakfast, 1·63 per mille; three hours after breakfast, 1·21 per mille.

August 23: Anxious. Before breakfast, 1·30 per mille; three-quarters of an hour after breakfast, 1·71 per mille; one and a half hours after breakfast, 1·40 per mille; two and a quarter hours after breakfast, 1·18 per mille; three hours after breakfast, 1·11 per mille.

September 12: Still depressed, but less anxious. Before breakfast, 1·17 per mille; three-quarters of an hour after breakfast, 1·49 per mille; one and a half hours after breakfast, 1·22 per mille; two and a quarter hours after breakfast, 1·16 per mille; three hours after breakfast, 1·13 per mille.

(No muscular exertion during the days of examination.) Left hospital unrecovered.

No. 19.—B. J. D., male, aged 58. Family history negative. Patient had one attack of melancholia before. This time: depression, anxiety, motor inhibition, delusion of having sinned, of poverty, attempt at suicide. No hallucinations, no dementia, no bodily changes.

Blood-sugar.

December 27, 1918: Depression, motor inhibition, no marked anxiety. Before breakfast, 1·24 per mille; three-quarters of an hour after breakfast, 1·56 per mille; one and a half hours after breakfast, 1·51 per mille; two and a quarter hours after breakfast, 1·43 per mille.

January 7, 1919: *status idem.* Before breakfast, 1·02 per mille: three-quarters of an hour after breakfast, 1·67 per mille; one and a half hours after breakfast, 1·44 per mille; two and a quarter hours after breakfast, 1·27 per mille.

February 27: No longer depressed, nearly totally recovered. Before breakfast, 1·0 per mille; three-quarters of an hour after breakfast, 1·29 per mille; one and a half hours after breakfast, 0·99 per mille; two and a quarter hours after breakfast, 0·94 per mille.

No. 20.—H. B. B., female, aged 56. One melancholic attack ten years ago. This time: depression, motor inhibition, anxiety, depressive delusions, no dementia, no bodily changes.

Blood-sugar.

August 15, 1918: Still depressed; no longer anxious; 1·04 per mille.

August 17: Still depressed; no longer anxious; 0·78 per mille.

August 19: Still depressed; no longer anxious; 0·93 per mille.

August 20: Still depressed; no longer anxious; 1·13 per mille.

November 18: Recovering. Before breakfast, 1·01 per mille; three-quarters of an hour after breakfast, 1·19 per mille; one and a half hours after breakfast, 1·26 per mille; two and a quarter hours after breakfast, 0·95 per mille: three hours after breakfast, 0·99 per mille. Left hospital nearly recovered.

No. 21.—D. O. — D., female, aged 34. Suffered from melancholia twice before. This time: depression, motor inhibition, anxiety, no delusions, no dementia; no bodily changes. (Patient was recovering from illness on entering the hospital.)

Blood-sugar.

October 11, 1918: Quite restored to normal psychical state. Before breakfast, 1·04 per mille; three-quarters of an hour after breakfast, 1·21 per mille; one and a half hours after breakfast, 0·79 per mille; two and a quarter hours after breakfast, 0·78 per mille; three hours after breakfast, 0·80 per mille

No.	Name	Age	Sex	Before breakfast	¾ hour after breakfast	1½ hours after breakfast	2¼ hours after breakfast	3 hours after breakfast	Highest amount
				Per mille	Per mille	Per mille	Per mille	Per mille	Per mille
1	P. de V. ..	65	M.	1·12	1·23	1·22	1·15	1·22	1·36
2	J. R.	66	M.	1·25	1·50	1·20	1·10	—	1·83
3	H. J. P. K. ..	43	F.	1·10	1·42	1·14	—	0·91	1·42
4	v. W.	60	F.	0·93	1·41	—	1·22	0·96	1·41
5	G. D. H. ..	23	F.	1·04	1·55	1·38	1 09	0·98	1·69
6	B. F. de L. ..	41	F.	1·26	1·89	1·64	1·20	—	1·96
7	M. M.	48	F.	1·24	1·39	1·37	1·39	1·43	1·43
8	D. L.	45	M.	1·15	1·76	1·47	1·25	—	1·90
9	A. v. d. W. ..	38	F.	0·97	1·24	1·18	0·92	—	1·34
10	A. M. J. M. H.	41	F.	1·05	1·43	1·36	1·28	1·23	1·53
11	C. K. B. ..	50	F.	1·31	1·66	1·33	1·25	—	1·70
12	J. M.	34	F.	1·01	1·44	1·22	1·00	1·00	1·51
13	E. de H. ..	44	M.	1·04	1·56	1·21	0·97	0·84	1·61
14	F. K.	68	M.	1·13	1·57	1·52	1·39	1·24	1·57
15	J. P. L. S. ..	51	F.	1·33	1·74	1·69	1·53	—	1·82
16	L. S. B. ..	61	F.	1·03	1·62	1·62	1·15	—	1·73
17	M. D. O. ..	47	F.	1·01	1·57	1·50	1·10	1·14	1·77
18	L. M. v. W. ..	38	F.	1·11	1·79	1·64	1·40	1·16	1·88
19	B. J. D. ..	58	M.	1·13	1·61	1·47	1·35	—	1·67
	Average			1·12	1·55	1·39	1·21	1·10	1·64
	Average of patients with marked anxiety (Nos. 2, 5, 8, 11, 16, 17, 18)			1·13	1·63	1·45	1·19	—	1·79
	Average of patients without obvious anxiety (Nos. 1, 3, 4, 9, 10, 12, 13)			1·03	1·39	1·22	1·03	—	1·45
	Average of patients aged above 50 (Nos. 1, 2, 4, 11, 14, 19)			1·13	1·50	1·39	1·24	—	1·59

As may be seen from the table (p. 238),[1] blood-sugar is much increased in melancholia. Firstly, the amount before breakfast is often too high, the average being 1·12 per mille. This is conformable to the results of Heidema, who found the average of 1·11 per mille, whereas Wigert (1919)[2] found an average of 0·95 per mille in his fourteen cases of melancholia. If we take 1·10 per mille as the limit for normal persons before breakfast, of my nineteen cases, nine had surpassed that number; of Heidema's seventeen cases, ten showed an amount above 1·10, whereas only two of Wigert's cases had surpassed the limit. As the result of these researches, we may say that spontaneous hyperglycæmia is often absent in melancholia, though very likely it is also more often present here than in other psychical diseases. Wigert also examined the alimentary hyperglycæmia, and here his results are in good accordance with mine. He gave 80 grm. glycose and compared his results with those of Jacobsen (1913, No. 97) in normal persons after the same dose of glycose; with normal individuals the amount of 1·40 was surpassed in 56 per cent. of the cases; with the patients of Wigert, this was the case with 91 per cent. Glycosuria (after taking 80 grm. glycose) was found absent by Jacobson in 65 per cent.; with melancholia according to Wigert only in 17 per cent.

For further details I refer to the paper of Wigert: these numbers may suffice to show the perfect harmony between the results of Wigert and my own; whereas the amount before breakfast is very inconstantly increased, there is in melancholia a fairly constant alimentary hyperglycæmia, as well after starch as after glycose.

To what factor must this increased blood-sugar be attributed? That this depends on the illness itself is clear, not only from the long-known fact that glycosuria appears and disappears with the depression, but also from my researches, as I was able to examine the Cases 11, 14, 15, and 19 while they were recovering; in these cases the amount of blood-sugar had decreased on recovery. To many of Wigert's and my own patients, opium was given. But opium has, according to af Klercker (1914-15), a decreasing influence on the amount of blood-sugar, which he ascribes to its inhibitory power on the peristalsis of

[1] In this table the blood-sugar amounts noted for each patient represent the averages of the percentages found by the several examinations on the height of the disease.

[2] I have taken the average of his several examinations of each patient, and again the average of these numbers. They have great value on account of the large number of examinations.

the stomach. At any rate, my results cannot be increased by gastro-intestinal movement; the weak intestinal peristalsis in melancholia (even without opium) can only have a decreasing influence on the blood-sugar, as the sugar of the food is more slowly absorbed. Chloral hydrate, which, according to Jacobsen (1913 [96]), may cause an increase of blood-sugar, was only occasionally given to my patients; moreover, it was never prescribed on the days of examination.

That the age, which might be of influence according to Laudenheimer and Ehrenberg (see p. 220), does not make any difference is shown by the percentage of the several patients aged 50 or above (see table, p. 238). Wigert mentions the bodily weight as a factor which might influence the amount of blood-sugar. The increase of blood-sugar would be inversely proportional to the weight. I do not know whether any experiments have been made which would justify this supposition, but in this form it seems to be highly improbable. According to Scott (1914), the weight is probably without special influence upon the concentration of sugar in the blood; it decreases, however, as the amount of blood drawn per kilo of body weight of the animal increases. Sufficient data have not been obtained to establish the mathematical expression for this relation ([160] p. 306). Wigert suggests still another possibility. According to Bang, sugar given to animals after hunger leads to a higher amount than when they are well fed; now melancholics are often under-fed, and this fact might partly explain the high blood-sugar after taking glycose. Of course I do not know the weight of my patients before admission, but I am sure that most of them are not under-fed after a long stay in hospital, as great care is taken to feed them regularly. This condition cannot be the reason of the increase of blood-sugar in my cases. Moreover, it cannot explain the several cases of high amounts before breakfast.

The theory of Raimann will be discussed in the following chapter on mania.

In my opinion only one satisfactory answer to the question can be given; *it is the emotional state that is responsible for the high amount of blood-sugar*. First of all, this supposition makes us understand the bad influence of emotions on saccharine diabetes; it is in agreement with the occurrence of emotional glycosuria in animals and man and to the fact that also of the other psychoses it is the emotional cases that show glycosuria, as was found by Mita for hysteria and neurasthenia; by von Jaksch (1895), Arndt (1897), and Horn (1915), for traumatic neuroses; by Goodhart for neurasthenia; by Travaglino (1911) for

catatonia and dementia senilis; by Strauss (1900) for imbecility; by Schultze and Knauer for dementia præcox, for imbecility and for dementia paralytica. [See also Case 7 (p. 227), of my patients suffering from dementia paralytica, and of epilepsy, Case 1 (p. 228)].

Blood-sugar in Neurasthenia and Psychasthenia.

It is here the place to mention four cases of true neurasthenia and psychasthenia without any bodily abnormalities, which I examined on several occasions.

No. 1.—K., male, aged 47, neurasthenic nervous person, but calm during examination: 1·04 per mille, 1·35 per mille, 1·00 per mille, 0·91 per mille, 0·90 per mille.[1]

No. 2.—H., male, aged 44; had suffered from psychasthenia for several years; quite calm during first examination: 1·09 per mille, 1·08 per mille, 1·09 per mille, 0·95 per mille, 1·02 per mille. Anxious and excited during second examination because of imperative ideas (phobias): 1·02 per mille, 1·50 per mille, 1·13 per mille, 0·92 per mille, 0·87 per mille.

No. 3.—D., male, aged 43, typical neurasthenic; made a very emotional impression that morning four hours before breakfast: 1·47 per mille.

No. 4.—E., male, aged 36 psychasthenia, agitation, imperative ideas, pulse 132. Two hours after breakfast (two rusks and one cup of milk): 1·87 per mille.

These results correspond quite well with those of the above-mentioned writers and show plainly the undeniable fact that emotions cause hyperglycæmia in man. I will add to these cases the account of six cases of *confusional insanity*. At first I intended to give a full survey of the emotional state and its reactions in this illness, as I have examined twenty cases. But afterwards the matter seemed to me to be too intricate to insert its detailed discussion into this paper; for it is still an open question whether amentia without palpable cause really exists (*vide* e.g., Stransky, 1904-06; Kraepelin, 1910), or whether confusional insanity after psychical or bodily traumata, after infectious diseases, or after delivery, &c., must be grouped together or ought to remain separated. Therefore I only took some representatives of the various groups merely to demonstrate the influence of emotion on the blood-sugar, also in this form of insanity. For convenience' sake I have called them all amentia.

[1] The numbers indicate the blood-sugar before breakfast, three-quarters of an hour after breakfast, &c.

Blood-sugar in Amentia.

No. 1.—H. T. G., female, aged 41. Husband died on November 16, 1918; patient became depressed in the last days of her husband's life; after his death, incoherence, excitement, hallucinations, laughing and singing alternately. No bodily changes, no fever.

Blood-sugar.

December 2, 1918: Exaltation, talking incessantly. Before breakfast, 1·04 per mille; three-quarters of an hour after breakfast, 1·13 per mille; one and a half hours after breakfast, 1·51 per mille; two and a quarter hours after breakfast, 1·35 per mille.

December 10: Same psychical state. Talking incessantly. Before breakfast, 1·30 per mille; three-quarters of an hour after breakfast, 1·24 per mille; one and a half hours after breakfast, 1·40 per mille; two and a quarter hours after breakfast, 1·31 per mille.

January 29, 1919: Much calmer. Before breakfast, 0·96 per mille; three-quarters of an hour after breakfast, 1·33 per mille; one and a half hours after breakfast, 1·34 per mille; two and a quarter hours after breakfast, 0·91 per mille.

No. 2.—R. v. D., female, aged 48, Jewess. Became incoherent after operation; then followed erysipelas; she recovered from both, but the insanity became worse; incoherence, illusions and hallucinations, disorientation, motor excitement: very marked exaltation; sometimes anxious and weeping: at other times singing; temperature a little raised (cystitis). No other bodily changes. Temperature in the mornings always normal, in the evenings sometimes normal, often a little raised.

Blood-sugar.

August 21, 1918: Before breakfast, 1·07 per mille.

August 22: Before breakfast, 1·66 per mille.

August 23: Before breakfast, 1·28 per mille.

August 24: Before breakfast, 1·11 per mille.

August 26: Before breakfast, 1·20 per mille; temperature normal in the morning, sub-febrile in the evening; patient alternately calm and excited; orientation much better; sometimes still hallucinations.

August 28: Before breakfast, 1·08 per mille; fifty minutes after breakfast, 1·44 per mille; one hour fifty minutes after breakfast, 1·71 per mille; two hours forty minutes after breakfast, 1·26 per mille.

August 29: Before breakfast, 1·20 per mille; three-quarters of an hour after breakfast, 1·48 per mille; one and a half hours after breakfast, 1·77 per mille; two and a quarter hours after breakfast, 1·44 per mille; three hours after breakfast, 1·26 per mille.

September 26: Recovered from illness, psychical state quite normal again. Before breakfast, 0·81 per mille; three-quarters of an hour after breakfast,

1·08 per mille; one and a half hours after breakfast, 1·04 per mille; two and a quarter hours after breakfast, 1·08 per mille; three hours after breakfast, 0·87 per mille.

No. 3.—J. P. G., female, aged 42. Family history negative; ten weeks before admission, eighth delivery without complications, seven weeks afterwards patient fell ill (never insane before); began with delusions and anxiety, afterwards exaltation; total incoherence; anxiety; complete disorientation. No bodily changes.

Blood-sugar.

November 1, 1918: Incoherent, sometimes anxious. Before breakfast, 1·39 per mille; three-quarters of an hour after breakfast, 1·64 per mille; one and a half hours after breakfast, 1·71 per mille; two and a quarter hours after breakfast, 1·69 per mille.

November 15: Quite recovered, normal psychical state. Before breakfast, 1·04 per mille; three-quarters of an hour after breakfast, 1·07 per mille; one and a half hours after breakfast, 1·22 per mille; two hours and a quarter hours after breakfast, 1·18 per mille.

No. 4.—A. V. E., female, aged 28 (puerperal insanity). No insanity before or during pregnancy; difficult labour; four days after delivery she became depressed; afterwards incoherent, very excited; in hospital: stupor, incoherence, sometimes exaltation.

Blood-sugar.

August 26: Calm, but incoherent: temperature normal. Before breakfast, 1·24 per mille.

August 28: The same. Before breakfast, 1·04 per mille.

August 29: The same. Before breakfast, 2·09 per mille (very angry with one of the nurses); three-quarters of an hour after breakfast, 1.52 per mille; one and a half hours after breakfast, 1·70 per mille; two and a quarter hours before breakfast, 1·49 per mille; three hours after breakfast, 1·39 per mille.

August 30: Before breakfast, 1·23 per mille; three-quarters of an hour after breakfast, 1·54 per mille; one and a half hours after breakfast, 1·54 per mille; two and a quarter hours after breakfast, 1·75 per mille; three hours after breakfast, 1·55 per mille.

October 7: Patient has greatly changed; no stupor; no exaltation or anxiety; but garrulity; childishness; tendency to make stupid jokes.

Before breakfast, 0·90 per mille; three-quarters of an hour after breakfast, 1·45 per mille; one and a half hour after breakfast, 1·46 per mille; two and a quarter hours after breakfast, 1·15 per mille; three hours after breakfast, 0·98 per mille.

November 15, 1918: Much better, is going to leave hospital: not quite recovered; still somewhat childish. Before breakfast, 0·84 per mille; three-quarters of an hour after breakfast, 1·33 per mille; one and a half hour, 1·38 per mille; two and a quarter hours after breakfast, 1·22 per mille.

No. 5.—H., female, aged 37. Family history negative; patient suffered once before from an attack of amentia after pneumonia. This time Spanish influenza; after fever had left, incoherence, disorientation, at first more stuporous, afterwards excited, anxious, totally incoherent. In hospital: incoherence; exaltation; extreme emotional state; hallucinations; anxiety. No bodily changes, no fever.

Blood-sugar.

November 16, 1918: Incoherent; very emotional; anxious; dilated pupils; high colour; rapid pulse; no motor excitement. Before breakfast, 1·19 per mille; three-quarters of an hour after breakfast, 2·04 per mille; one and a half hours after breakfast, 1·82 per mille: two and a quarter hours after breakfast, 1·12 per mille.

December 21: Much calmer; much less emotional; no colour; still some incoherence. Before breakfast, 1·28 per mille; three-quarters of an hour after breakfast, 1·46 per mille; one and a half hours after breakfast, 1·35 per mille; two and a quarter hours after breakfast, 1·12 per mille. Left hospital nearly recovered.

No. 6.—S., female, aged 56.—Family history negative. Patient never had any bodily or mental diseases. Caught Spanish influenza at the end of October. Became insane directly after influenza; confusion; incoherence; hallucinations; motor excitement; spitting; shouting; very restive; refuses to speak and to eat. No longer bodily changes. Temperature normal again.

Blood-sugar.

November 28, 1918: For psychical symptoms, see above. Before breakfast, 0·98 per mille; three-quarters of an hour after breakfast, 1·54 per mille; one and a half hours after breakfast, 1·76 per mille; two and a quarter hours after breakfast, 1·40 per mille.

December 14: Convalescent, much calmer. Before breakfast, 1·24 per mille; three-quarters of an hour after breakfast, 1·24 per mille; one and a half hours after breakfast, 1·34 per mille; two and a quarter hours after breakfast, 1·41 per mille.

Left hospital nearly recovered.

As may be seen from these reports, the blood-sugar is very much increased when the patients are in a highly emotional state, whereas it decreases on recovery. In the first four cases this increase cannot be ascribed to any bodily changes, as the reason of the illness had ceased to exist months ago, and no bodily abnormalities had remained. In the cases of amentia after Spanish influenza, one might think of toxins, which still were circulating and disturbing the metabolism; this, however, is very improbable, as other mental disorders after influenza, without excitement, in which according to that supposition the same

toxins must be present, do not show that high increase of blood-sugar (cf. Case 2, T. B., on p. 249).

The influence of anxiety is very striking, especially with melancholia, as may be seen from the table (p. 238); the amount of blood-sugar before breakfast in anxious patients is the same as the general average, but the increase after breakfast is greater, the curve being steeper. If we compare these amounts with those of patients without obvious anxiety, the difference is still clearer.[1] Of course the greatest difference exists between the averages of the highest amounts of anxious and not anxious patients. Therefore I take it for granted that anxiety plays an important part in melancholic hyperglycæmia; the only question which remains to be answered is whether blood-sugar is also increased in simple depression, without marked anxiety. First I may draw attention to the fact that anxiety in any degree is very seldom absent in true melancholia; one of the characteristics of pathological depression is that it nearly always shows a tinge of anxiety or agitation; I hardly remember a case where that symptom was wanting in the history and present state, if we look for it in all its variations, from pathognomonic restlessness and agitation to the highest precordial distress. How often we may find even in stupor, when all emotion seems to be extinguished, that anxiety was merely slumbering. That there may be still another reason why depression itself is associated with an increase of blood-sugar I hope to make clear in the second chapter.[2]

Blood-sugar in Mania.

We have but little information about the amount of blood-sugar in mania, as the illness is doubtless rare. Schultze and Knauer examined six cases, Raimann four, and Heidema three cases. My material, as regards true mania, is also rather poor, but the Spanish influenza has furnished the hospital with so many psychoses, of which several showed typical maniacal symptoms, that a discussion may be justified. Schultze and Knauer found no glycosuria in their cases except occasionally, when the patients were recalcitrant or excited. In accordance Raimann found a high assimilation-power in mania. Heidema, however, found a rather high amount in his three cases: 1·13, 1·28, and 1·60 per mille before breakfast.

[1] In the three cases of recovered melancholics I examined, hyperglycæmia appeared to be absent.

[2] After I had finished the table (p. 238), I examined eight more patients suffering from melancholia. The results formed a complete confirmation of those laid down in the table; all these patients, without any exception, showed a marked hyperglycæmia.

Now in mania it is difficult to answer our question; in the first place the motor excitement, if extreme, may cause a decrease of blood-sugar, whereas it may cause an increase if the movements are moderate, and can be compared with normal bodily labour (cf. p. 220). In the second place, a sufferer from mania may be examined in very different moods, especially different in regard to the question we are discussing. Whereas the melancholiac is full of his grief, and only more or less agitated, but always in an emotional state, the maniacal patient is optimistic, cheerful, talkative, then again highly excited, nearly incoherent, or in a state of severe excitement and unwilling towards the examiner. I believe this point to be of grave importance in judging the results of the various authors.[1]

Schultze and Knauer found glycosuria with maniacal patients who were excited and resistive; of the four cases of Raimann, three had nearly recovered, of the remaining case we find noted that she was cheerful and erotic. Heidema's three cases were all of the severe type, highly excited, suffering from want of sleep, shouting and destructive.

I shall now discuss my own cases. I must once again draw the attention to Case 15 in my discussion of melancholia, where a melancholic stage suddenly changed into a maniacal one, and then showed a much lower amount of blood-sugar.

No. 1.—A. L. B., female, aged 29. Suffered from mania once before. This time: exaltation; accelerated flow of thoughts; motor excitement; easily distracted; always joking; not markedly emotional; no dementia; no bodily abnormalities; short depressive period before mania.

Blood-sugar.

October 3, 1918: For psychical symptoms *vide supra;* no extreme movements, for patient is tied to the bed. Before breakfast, 0·86 per mille; three-quarters of an hour after breakfast, 1·36 per mille; one and a half hours after breakfast, 1·33 per mille; two and a quarter hours after breakfast, failure; three hours after breakfast, 1·06 per mille.

October 22: Same condition; always joking; no real excitement. Before breakfast, 0·96 per mille; three-quarters of an hour after breakfast, 1·19 per mille; one and a half hours after breakfast, 1·07 per mille; two and a quarter hours after breakfast, 0·77 per mille; three hours after breakfast, 0·79 per mille.

Left hospital unrecovered.

[1] I must, however, call to mind once more the small number of the patients examined, and I do not believe that we are justified in considering our opinion of the carbo-hydrate metabolism in mania a final one.

No. 2.—P. S. H., male, aged 36. Had an attack of mania twice before, recovered completely each time. Now: exaltation; accelerated flow of ideas; no marked motor excitement; easily distracted; delusions of maniacal nature; stereotyped movements; always in high spirits; no bodily changes.

Blood-sugar.

October 23, 1918: Psychical state *vide supra*. Before breakfast, 1·16 per mille; three-quarters of an hour after breakfast, 1·44 per mille; one and a half hours after breakfast, 1·88 per mille; two and a quarter hours after breakfast, 1·26 per mille; three hours after breakfast, 1·24 per mille.

November 5: same psychical state. Before breakfast, failure; three-quarters of an hour after breakfast, 1·32 per mille; one and a half hours after breakfast, 1·51 per mille; two and a quarter hours after breakfast, 1·27 per mille; three hours after breakfast, 1·17 per mille.

February 3, 1919: Much calmer; still in merry mood; joking, but without real excitement. Before breakfast, 1·03 per mille; three-quarters of an hour after breakfast, 1·39 per mille; one and a half hours after breakfast, 1·09 per mille; two and a quarter hours after breakfast, 1·09 per mille.

No. 3.—M. O., male, aged 16. Family history negative; abnormal for a few weeks; talkative; sleepless; wandering about in the night; borrowed 50 guilders and spent it in cafés. In hospital he shows all the symptoms of true mania: exaltation; motor excitement; accelerated flow of thoughts; easily distracted; he is very destructive; singing; shouting; spitting; very resistive; often isolated or in continuous bath; no symptoms at all of dementia præcox; no bodily changes; no dementia.

Blood-sugar.

November 30, 1918: Showing all the symptoms mentioned above. Before breakfast, 1·00 per mille; three-quarters of an hour after breakfast, 1·24 per mille; one and a half hours after breakfast, 1·36 per mille; two and a quarter hours after breakfast, 1·57 per mille.

March 1, 1919: Exaltation; resisting puncture; furious; afterwards calmer. Before breakfast, 0·91 per mille; three-quarters of an hour after breakfast, 1·51 per mille; one and a half hours after breakfast, 1·36 per mille; two and a quarter hours after breakfast, 0·91 per mille.

March 21: Much calmer during last week; optimistic; cheerful; talkative; easily distracted but not excited. Before breakfast, 0·92 per mille; three-quarters of an hour after breakfast, 1·00 per mille; one and a half hours after breakfast, 1·11 per mille; two and a quarter hours after breakfast, 0·91 per mille.

No. 4.—H. B. d. L., male, aged 28. Family history negative. In 1912 short depression, and again during mobilization 1914; wept much at the death of his mother, who died in 1912. In 1917 maniacal stage, followed by a depressive period. In August, 1918, again maniacal period; motor excitement; very talkative; sometimes incoherent; singing; wandering about at night;

giving away his money: irritable; often fighting. In hospital much calmer; talkative; optimistic; singing; not obedient to nurses; meddlesome.

Blood-sugar.

February 1, 1919: Symptoms mentioned above. Before breakfast, 1·09 per mille: three-quarters of an hour after breakfast, 1·01 per mille; one and a half hours after breakfast, 0·98 per mille; two and a quarter hours after breakfast, 0·95 per mille.

February 6: Same psychical state. Before breakfast, 0·83 per mille; three-quarters of an hour after breakfast, 1·20 per mille; one and a half hours after breakfast, 0·94 per mille; two and a quarter hours after breakfast, 1·05 per mille.

No. 5.—A. G., female, aged 28. Family history negative. Became abnormal in the autumn of 1918, excited, talkative, sleepless, alternately depressed and optimistic. After a short period of depression, she became maniacal in the beginning of 1919: exaltation, accelerated flow of thoughts, sometimes incoherence; was irritable, neglected her work, could not remain at home any longer. In hospital, excited, meddlesome, talkative, very irritable, resistive, changing her mood every moment, often weeping, but usually showing the maniacal symptoms. No bodily changes, no symptoms of dementia præcox.

Blood-sugar.

January 17, 1919: Exaltation. Symptoms: see above. Before breakfast, 1·67 per mille; three-quarters of an hour after breakfast, 1·90 per mille; one and a half hours after breakfast, 1·42 per mille; two and a quarter hours after breakfast, 1·11 per mille.

February 5.—Still the same maniacal symptoms, mixed with melancholic ones; weeping this morning, afterwards optimistic, cheerful. Before breakfast, 1·24 per mille; three-quarters of an hour after breakfast, 1·72 per mille; one and a half hours after breakfast, 1·39 per mille; two and a quarter hours after breakfast, 1·03 per mille.

As may be seen from these reports, the results with mania are very variable. In my cases, too, it appears that a simple cheerful optimistic mood usually does not give a high amount of blood-sugar, as may be seen from Case 4; Case 1 was not really emotional. Case 2, on becoming calmer, and being only jocular and optimistic, showed only a slight increase of blood-sugar, while the amount was very high at the climax of the illness. Case 3 was excited and resisting, and excited during the beginning of the second examination; when the patient came to a calmer stage of the illness, and emotion had gone, the blood-sugar was normal again. Case 5 was very emotional; this patient sometimes showed the mixed form of mania depressiva, though as

a rule she was maniacal; this state was analogous to that of Case 1 of the amentia group, in which there was also that instability of mood with the strong tendency to optimism and highly increased blood-sugar.

When we now examine the results with *mania or mania-like psychoses after Spanish influenza*, we see this conception affirmed.

No. 1.—G. t. H., female, aged 19. Quite normal till November 13, 1918: influenza; on November 27 she became excited, incoherent. On admittance: exaltation, motor excitement, incoherence, fever.

Blood-sugar.

November 3, 1918: Motor excitement, accelerated flow of thoughts, no fever. Before breakfast, 0·93 per mille; three-quarters of an hour after breakfast, 1·41 per mille; one and a half hours after breakfast, 1·36 per mille; two and a quarter hours after breakfast, 1·04 per mille.

March 20, 1919: Recovered. Before breakfast, 0·74 per mille; three-quarters of an hour after breakfast, 1·08 per mille; one and a half hours after breakfast, 0·80 per mille; two and a quarter hours after breakfast, 0·75 per mille.

No. 2.—T. B., female, aged 28. Father had suffered from melancholia, grandmother died insane, one aunt suffered from epilepsy, another from imbecility. Became incoherent during attack of influenza and pneumonia (before influenza always quite normal). Remained abnormal after recovery from pneumonia. On admission: Talkative, talking nonsense (but knowing this), very erotic, without shame, optimistic, cheerful, no exaltation, no great emotions, no incoherence, no delusions, no hallucinations. Remained in this state for months. Left hospital quite recovered.

Blood-sugar.

December 12, 1918: Symptoms: see above. Before breakfast, 1·06 per mille; three-quarters of an hour after breakfast, 1·20 per mille; one and a half hours after breakfast, 1·24 per mille; two and a quarter hours after breakfast, 1·07 per mille.

December 19: Same psychical state. Before breakfast, 0·84 per mille; three-quarters of an hour after breakfast, 1·22 per mille; one and a half hours after breakfast, 1·29 per mille; two and a quarter hours after breakfast, 1·20 per mille.

January 4, 1919: Same psychical state. Before breakfast, 1·05 per mille; three-quarters of an hour after breakfast, 1·20 per mille; one and a half hours after breakfast, 0·99 per mille; two and a quarter hours after breakfast, 1·16 per mille.

No. 3.—R. B., male, aged 34. Family history negative. Quite normal before influenza. Caught Spanish influenza three weeks before admission. Quite recovered ten days afterwards. Some days after, however, he became

excited, talkative, had delusions of expansive character. In hospital: exaltation, accelerated flow of thoughts, motor excitement, easily distracted, no incoherence, no disorientation, no hallucinations, cheerful, optimistic, singing, expansive delusions, no fever, no bodily changes.

Blood-sugar.

November 16, 1918: For symptoms see above. Before breakfast, 1·01 per mille; three-quarters of an hour after breakfast, 1·35 per mille; one and a half hours after breakfast, 1·25 per mille; two and a quarter hours after breakfast, 1·17 per mille.

November 30: Same psychical state, not very emotional. Before breakfast, 0·98 per mille; three-quarters of an hour after breakfast, 1·25 per mille; one and a half hours after breakfast, 1·27 per mille; two and a quarter hours after breakfast, 1·29 per mille.

No. 4.—J. v. D., male, aged 42. Family history negative. Quite normal until Spanish influenza in the beginning of November. After recovery he worked again for some days, then became abnormal: talkative, excited, restless, talking nonsense and wandering about; no incoherence. In hospital: exaltation, motor excitement, meddlesome, distractibility, no disorientation, no incoherence, no delusions, cheerful, not irritable, never angry, no fever, no bodily changes.

Blood-sugar.

December 2, 1918: Slight exaltation; no motor excitement. Before breakfast, 1·27 per mille; three-quarters of an hour after breakfast, 1·25 per mille; one and a half hours after breakfast, 1·30 per mille; two and a quarter hours after breakfast, 1·21 per mille.

December 20: Still slight exaltation, optimistic. Before breakfast, 1·25 per mille; three-quarters of an hour after breakfast, 1·60 per mille; one and a half hours after breakfast, 1·10 per mille; two and a quarter hours after breakfast, 1·03 per mille.

No. 5.—A. G., female, aged 22. Family history negative. Emotional character, but no abnormalities before Spanish influenza in the beginning of November. Soon after recovery she became irritable, excited, talkative, sometimes destructive. In hospital: very much excited; sudden changes of mood; as a rule cheerful, making childish jokes motor excitement, easily distracted.

Blood-sugar.

January 17, 1919: Strong exaltation; puncture could only be performed twice. Before breakfast, 1·15 per mille; three-quarters of an hour after breakfast, 1·44 per mille.

February 5: Still excited; same psychical symptoms. Before breakfast, 1·04 per mille; three-quarters of an hour after breakfast, 1·48 per mille;

one and a half hours after breakfast, 1·36 per mille; two and a quarter hours after breakfast, 1·32 per mille.

No. 6.—F. K., female, aged 23. Family history negative. Patient was quite normal until she caught Spanish influenza on November 20, 1918, together with pneumonia. Left bed December 8. On the following day she became excited; talkative. Became more excited every day; talking, singing, incoherent, now and then hallucinations. In hospital: exaltation, accelerated flow of thoughts, no marked motor excitement, optimistic, singing, rhyming, incoherent; no bodily changes, no fever.

Blood-sugar.

December 13, 1918: Marked exaltation, incoherence. Before breakfast, 1·25 per mille; three-quarters of an hour after breakfast, 1·71 per mille; one and a half hours after breakfast, 1·70 per mille; two and a quarter hours after breakfast, 1·33 per mille.

December 19: Same psychical state; maniacal. Before breakfast, 1·14 per mille; three-quarters of an hour after breakfast, 1·62 per mille; one and a half hours after breakfast, 1·32 per mille; two and a quarter hours after breakfast, 1·24 per mille. Left hospital quite recovered. I omitted to examine her blood-sugar then.

The Cases 2, 3 and 4 were not of a highly emotional nature, the emotions being more superficial and transitory, especially in Case 2. The other cases showed more real exaltation. I have, however, no explanation to give for the high amount in the second examination of Case 4.

We may therefore conclude:—

(1) That the amount of blood-sugar in mania and maniacal states often shows a marked increase.

(2) That this increase is highest in real emotional states, such as may occur in mania at its climax, when we almost see the picture of acute delirium and the patient is destructive and very irritable.

(3) That it is smaller, though still present, in the milder form of mania, and

(4) Absent in those hypomaniacal forms, in which the patient is only optimistic, cheerful and inclined to be jocular.

Of course, this conception is quite opposed to that of Raimann. That author, as has been mentioned, found a low assimilation power in melancholia and a high one in mania. He attributes this fact to the inhibition peculiar to melancholia which would also affect the combustion of sugar. Now he examined four cases only of mania, of which at any rate three were convalescent; of the fourth case we

do not know whether it was a mild or a severe form. It was therefore impossible for him to judge of the condition in a high emotional state of mania. As for his explanation of the increase in melancholia, this cannot be the correct one. Firstly, it would not explain why the highest increase of blood-sugar was principally found in the patients with marked anxiety and agitation, not only in melancholia, but also in neurasthenia, psychasthenia, amentia, &c.; in these cases the metabolism should rather be accelerated than inhibited. Moreover, according to Bornstein (1908-10) and Grafe (1910), the respiratory quotient is not altered in melancholia, not even in stupor. In this respect there is a marked difference between the (emotional) stupor of melancholia and that of catatonia and paralytic insanity. As for the bodily reactions, melancholia and mania are apparently not so strongly contrasted as might be supposed from their psychical attitudes; this may be easier understood when we bear in mind that these reactions are dependent on, or at least parallel to, emotions, and that both, melancholia and mania, are based on an increased emotional state. That simple depression gives a constant increase of blood-sugar, whereas it is wanting in cheerfulness and optimism, will be explained in the second chapter.

Chapter II.—The Theories of Cannon and of Gaskell.

Looking for the deeper reason of this phenomenon, I have argued as follows: Hyperglycæmia in mental diseases is caused by strong emotions, especially those of fear and anger. It is the same emotions which cause glycosuria and hyperglycæmia in animals, and doubtless the phenomenon is the same in man and animals. For if ever a comparison is permitted between human pathology and animal experiments, it is here. Emotions, and especially those of fear and anger, are, phylogenetically speaking, old psychical processes, and are still provided with the old animal reactions; in anger and fear men become animals again. Therefore we must look for the solution of our problem in the animal world, where conditions are much simpler.

Cannon, in his article on emotional glycosuria (1911), was the first to suggest that hyperglycæmia might be useful to the animal, as it provides the muscles with a larger amount of sugar, which is combusted during the actions which may follow or accompany the emotions—i.e., in fight and flight. Then the question arose, whether the mobilization

of sugar would be the only consequence of this strong sympathetic impulse in emotion, due to the increase of epinephrin. *A priori* this seemed improbable, as both adrenalin and stimulation of the sympathetic nerve usually have a marked general effect. And, indeed, we find in the excited animal several signs of increased sympathetic stimulation, such as the increase of the heart rate, the widening of the pupils, and the erection of hairs. If Cannon's suggestion were true, it would be probable that not only the increase of blood-sugar, but also the other reactions, dependent on sympathetic stimulation, were useful to the animal in motion. In his splendid monograph on the "Bodily Changes in Pain, Hunger, Fear and Rage" (1918), Cannon has answered this question in the affirmative. However, before giving a short résumé of the principal contents of this publication, I must discuss in short Gaskell's last book, "The Involuntary Nervous System." The conception of the nervous system of this ingenious author seems to me to be of great value for our present problem.

Gaskell points out that the involuntary nervous system is built up on the same principle as the voluntary one; for his arguments I must refer to his publication. According to him, not only the voluntary but also the involuntary nervous system can be divided into a somatic and a splanchnic part. The somatic part is represented by the thoracico-lumbar outflow (sympathetic division); it supplies the heart and the blood-vessels throughout the entire body with motor fibres, and a system of involuntary muscles which possibly arose from a system of dermal muscles, situated exactly under the skin over the whole body ([74], p. 63). It is segmentally built, analogous to the voluntary somatic system which sends motor fibres to the muscles of trunk and extremities. The splanchnic part of the involuntary nervous system, consisting of the bulbar and the sacral outflow,[1] sends motor fibres to the muscles of the alimentary canal and its derivatives; similarly the splanchnic part of the voluntary system innervates only muscles of splanchnic or originally splanchnic function (fifth, seventh, tenth nerves).

According to Gaskell, this division of the nervous system would be more ancient than that into a voluntary and an involuntary part. For the original animal, from which, in his opinion, the vertebrate arose,

[1] "Autonomes System," of Eppinger and Hesz (1910); in this term the cranial outflow (n. oculomotorius) is included. Gaskell, however, considers the muscular system belonging to this (prosomatic) outflow as a separate group (op. cit., p. 66).

possessed a double segmentation throughout its whole length; a body segmentation and an appendage segmentation. The muscles of these two segmentations, derived originally in the segmented annelids from unstriped muscle, became in part striated and voluntary and partly remained unstriped. The unstriped muscles of the somatic segmentation form the sympathetic or epidermal musculature, and those of the appendage segmentation, the endodermal musculature . . ." (op. cit., p. 64). Sympathetic stimulation in emotions of fear and rage seemed to me much more easily comprehensible in the light of this conception; for it would only be in those conditions which led to the emotions in question, i.e., when the animal had to flee or to fight, that the whole somatic nervous system would come into action, not only the voluntary but also the involuntary system. All the energy of the animal would in moments of danger be withdrawn from the splanchnic system and would be thrown into the somatic system; the striking contrast which the two systems in their functions show would be easily understood. If Cannon's suppositions were right, they would mean a physiological affirmation of Gaskell's anatomical conception.

So two questions remain to be discussed:—

(1) Whether in emotions there really is a general sympathetic stimulation and whether all the resulting reactions are of use to the animal.

(2) Whether in mental disorders the same general sympathetic stimulation is found. For if the increase of blood-sugar has the same cause as it is supposed to have in animals, the parallel must possibly be followed to the end.

Cannon's Theory.

As I have mentioned, Cannon has already answered the first questions in the affirmative. He has ingeniously combined the various researches of himself and his collaborators, and it seems to me that his suggestion in 1911 has now become a solid theory. For the many interesting details, I must refer to his publication. I am, however, obliged to discuss the principal contents shortly, as this is necessary for the right comprehension of the bodily changes in melancholia.

First Cannon points out that emotions such as rage and fear are unfavourable for the normal secretion of the digestive juices and for the contraction of the stomach and intestines, i.e., for those functions that are dependent on stimulation of the splanchnic division of the

involuntary nervous system (nervus vagus). On the contrary, these emotions are accompanied by an increased stimulation of the sympathetic division, or, what is the same thing, by increased adrenal secretion. The effect resulting from this stimulation is of signal service to the animal, which must be able to run or fight in the conditions which had led to its emotional state. For Cannon has proved that not only the blood-sugar is increased, by which phenomenon the muscles are enabled to work more and longer, but also that increased adrenalin is an antidote to the effects of fatigue. Moreover, adrenalin has a stimulating effect on the heart and increases the blood-pressure. Now the vessels supplying a muscle dilate when the muscle becomes active; when many muscles are involved in activity, a widely spread dilatation might reduce the blood-pressure to the point at which there would be insufficient circulation in active organs. Therefore the rise of the general arterial pressure is a useful and even necessary compensation. Moreover, adrenalin has no effective influence on the vessels of the lungs and the brain and a vasodilator instead of vaso-constrictor action on the vessels of the heart. Thus the absolutely and immediate essential organs—the heart, the lungs, and the brain as well as its instruments, the skeletal muscles—are in times of excitement abundantly supplied with blood, taken from organs of less importance in critical moments (the intestinal organs) (see Cannon, op. cit., p. 107). The changes in the respiratory function caused by emotions are also favourable to great effort. The deep and rapid respiration which reduces the carbon-dioxide contents of the blood is accompanied by relaxation of the smooth muscles of the bronchioles resulting from increased adrenalin (Trendelenburg, 1912), so that the current of air may pass more freely. Lastly. adrenalin causes a rapid coagulation of the blood which may prevent a possible loss of blood of the fighting animal.

Sympathetic Reactions not discussed by Cannon.

Some effects of increased sympathetic stimulation[1] are not discussed in this monograph.

Erection of hairs.—First there is the erection of hairs; the musculi erectores pilorum are innervated by the sympathetic division, and they certainly come into action during emotions, such as rage and fear.

[1] In speaking of increased sympathetic stimulation, I also include increased adrenalin secretion, as those two always accompany each other and have the same effect (Elliot).

According to Darwin (1873) the aim is to terrify the adversary, and it is used by some animals in attack (dog), by others in defence (cat); this phenomenon is not only found in mammals, but also in the lower vertebrates (birds and reptiles).

Dilatation of the pupils.—Secondly there is a widening of the pupils in emotion, and this too is a well-known effect of sympathetic stimulation. Cannon only mentions the contracting power of the splanchnic (cranial) division as a proof of the conservative function of this part of the involuntary nervous system (protection of the retina from too intensive light). As widening of the pupil, however, is an active sympathetic phenomenon, according to our scheme we must look for its utility. There are two contrasting opinions about the dilatation of the pupil on sensory and psychical stimuli. Formerly it was the general opinion that these stimuli were conducted by the cervical sympathetic, and accordingly the dilatation of the pupil was called sympathetic reaction. Nowadays most authors seem to be of the opinion of Braunstein (1894) and Bechterew (1895), that the widening of the pupil, dependent on sensory and psychical stimuli, is caused by the inhibitory influence of the cortex cerebri on the tonus of the sphincter iridis (Bumke, 1904—Weiler, 1910). Now we know from the researches of Lewandowsky (1899), that injections of adrenalin cause a dilatation of the pupil. Langley (1901), Gaskell (1916), Elliot (1905) and Eppinger and Hesz (1910) state that both stimulation of the sympathetic and injection of adrenalin cause a dilatation of the pupil. Basing his view on the researches of Waymouth Reid (1895), Gaskell believes in the reciprocal innervation of the m. sphincter and the m. dilatator iridis by the third nerve and the sympathetic, the latter stimulating the m. radiator and inhibiting the sphincter pupillæ. We further know that emotions such as rage and fear cause an increase of adrenalin secretion (Cannon and de la Paz, 1911), and that the same emotions are accompanied by a dilatation of the pupil. In view of these facts, the supposition seems evident that emotion causes dilatation of the pupil by way of increased adrenalin secretion (probably together with a direct stimulation of the cervical sympathetic).

The arguments in favour of the theory of the inhibitory influence of the cortex are chiefly two:—

(1) When the tone of the sphincter iridis has disappeared, sensory and psychical reaction can no longer be obtained. This may be brought about by cutting the third nerve (Braunstein, 1894) or by preventing the light from exerting a stimulating influence on the sphincter tone.

For instance, if we shut the normal eye of a man or animal, whose optic nerve of the other side does not conduct the light, reflex-dilatation is no longer observed on the blind eye (Bechterew, 1883; Braunstein, 1894; Bach, 1908; Weiler, 1910). I want to presuppose first that the example given by Weiler, of tabes, with loss of light-reflex, and at the same time of the sensory-psychical reactions (Erb, Gowers, 1886, and others) is badly chosen, for in tabes the sympathetic system is very probably affected and therefore the loss of psychical reflexes does not prove anything with regard to sympathetic innervation. Gowers (1886) writes in his text-book on nervous diseases (p. 297): "The pupils are often very small (spinal miosis), especially when there is loss of the reflex-dilatation from the skin, a loss which seems to be accompanied by atony of the radiating fibres, supplied by the sympathetic. The pupils are not always small, they may be of medium size or beyond medium size, and then I have usually found that the skin-reflex can be obtained, although the light-reflex cannot." This observation stands rather for than against the sympathetic theory. But even if the conduction of the optic nerve is destroyed by another process, or if the n. oculomotorius is cut, the loss of reflex-dilatation does not go against the sympathetic theory. For in that case there is a hyperfunction of the sympathetic, which makes the pupils dilate, sometimes maximally; psychical and sensory stimuli cannot dilate it any further. Strong sensory stimuli, however, may even then cause a widening of the pupils, of which fact Weiler gives the following explanation: "Sensory and psychical stimuli act exclusively by means of a centre in the cortex cerebri; from this centre inhibitory fibres run to the nucleus of the third nerve. Now sensory stimuli, such as pain, follow the same route, but they act by means of the cervical sympathetic." In view of the strong analogy in the bodily changes, caused by pain and by emotions such as rage and fear, as proved by Cannon, this difference is very improbable. I may here mention the fact that all kinds of asphyxia (Bumke) cause dilatation of the pupils; this tallies very well with the researches of Cannon and Hoskins (1911), who found in asphyxia hypersecretion of epinephrin and increase of blood-sugar.

(2) The second argument against the sympathetic theory is the fact that extirpation, or paralysis of the cervical sympathetic, does not prevent reflex dilatation [Vulpian (1879), Bumke, Weiler]; but it must not be forgotten that the emotional stimulus still reaches the adrenal glands by the splanchnic nerves and that hypersecretion of adrenalin is not prevented. Moreover all authors agree that by extirpation of the

cervical sympathetic the dilatation reflex is altered, at least the reflex on sensory stimulation. I shall afterwards return to this point. The advocates of the cortex theory see an affirmation of their opinion in the researches of those authors—e.g., Braunstein, and Parsons (1901)—who found an inhibitory influence on stimulation of the cortex cerebri, which could be prevented by cutting the n. oculomotorius. Many other observers could not obtain these results in analogous experiments; the researches of Karplus and Kreidl (1909, 1910), on the contrary, proved that there is a cerebral localization of the sympathetic reflex and that this centre is indispensable for the dilatation of the pupil after stimulation of the sciatic nerve. All agree, however, in this point, that the reflex must be localized in the cerebrum above the pons Varolii. There seems to me to be much evidence for the opinion that with the reflex in question the centrifugal stimulus is conducted from the cerebrum to the sympathetic system, at the same time affecting the cervical sympathetic and the splanchnic. Stimulation of the first will give a nearly direct response to the original stimulus. Probably the same stimuli have an inhibitory influence on the tone of the sphincter pupillæ, according to the results of the above-mentioned observers, a view in perfect harmony with our ideas about the reciprocal innervation of the sphincter and the dilator iridis. Stimulation of the splanchnic, however, causes an increase of adrenalin in the blood and reaches the eye a little later. In this way a remarkable fact stated by former examiners may be easily understood. Vulpian had already found that the quick response of the pupil on stimulation of the n. ischiadicus changed into a slower one with a longer latent period after extirpation of the cervical sympathetic; the same was found by Braunstein. But then the direct sympathetic reaction was prevented; the remaining adrenalin reaction, however, could only appear after a certain latent period. The description given by Lewandowsky of the dilatation of the pupil on injection of adrenalin is analogous to those of Vulpian and Braunstein after excision of the cervical sympathetic. I may still draw attention to the fact, that after injections of adrenalin not only is the pupil widened, but protrusion of the bulb, widening of the palpebral fissure and retraction of the membrana nictitans also occur. Precisely the same effect was seen by Vulpian on stimulating the sciatic nerve after extirpation of the cervical sympathetic. Cannon and Hoskins (1911) could indeed show an increase of adrenalin after stimulation of the sciatic nerve. For the many questions which arise in connexion with the subject, one should see the works by Braunstein,

Bumke and Weiler. The scope of this article does not permit of a more detailed discussion. I was, however, obliged to discuss the principal facts in order to show that the emotional dilatation of the pupil does not form an exception to the theory of Cannon.

Darwin has already looked for the deeper reason of this reflex-dilatation. However, his remark that the brain is directly affected by the powerful emotion of fear and reacts on the pupils does not throw more light upon the question. Perhaps the solution of the problem must be looked for in the suggestion of Weiler, that in alarm the pupils of the animal were widened in order to expose the peripheral part of the retina to perception and to observe in this way surrounding objects over a wider range. I may add, that the widening of the palpebral fissure and the protrusion of the bulb would support this function of the pupil. I do not know, however, whether dilatation of the pupil causes an enlargement of the visual field in lower animals; it is probably not the case in man. Moreover, Weiler suggests that the dilatation reflex may be of use in moments of concentration of the attention (such as intensively hearing or thinking); for when we gaze in the distance we are not distracted by visual perception.

Salivary glands.—The innervation of the glandular structures offers some difficulties. Cannon discusses only the salivary glands and their innervation, together with the other parts of the digestive tract, by the cranial outflow of the involuntary nervous system (op. cit., pp. 9 and 31); doubtless the principal secretory function is bound to that division, as may be clear when we compare the copious liquid saliva obtained by stimulation of the chorda tympani with the tough, poorly secreted sympathetic saliva. Nevertheless there is evidence that these glands have also a sympathetic innervation, like the lachrymal glands, for Langley found in 1901 that injection of suprarenal extract caused secretion of all the salivary glands (op. cit., p. 126).[1] The explanation given by Gaskell of this double innervation is, that the two antagonistic systems supply with secretory fibres glandular structures which differ morphologically, part being epidermal glands and part being endodermal glands, the former being supplied by sympathetic fibres, while the latter would be supplied by connector nerves belonging to the bulbar group. In 1905, Elliot wrote that the question could "not be satisfactorily discussed until more is known of

[1] On the contrary, Oliver and Schäfer (1895) could not find any influence of adrenalin on the secretion of the salivary glands.

the double innervation of the salivary glands, and of the separate parts played by muscle in expelling the products of glandular activity." Eppinger and Hesz believe the salivary glands to have an autonomic innervation (the autonomic system of Eppinger and Hesz is the same as Gaskell's splanchnic division of the involuntary system); this opinion is based on the action of pilocarpine.

Be this double innervation as it may, it is certain that production of saliva would not be of use to an animal in rage or fear; but then it does not happen in these emotional states. For, as Cannon has pointed out, the mouth of an anxious person is dry. That the increase of adrenalin, caused by the same emotion does not give free salivary secretion may find its reason in the fact that, according to Langley (op. cit., p. 238), only large doses of the suprarenal extract are able to cause secretion of these glands. Small doses, though sufficient to cause a great rise of blood-pressure, may not have a secretory action; and indeed, the amount of adrenalin following physiological stimulation of the adrenal glands is, according to Osgood (quoted from Cannon)[1] very small.

Lachrymal glands.—According to Gaskell, a similar double innervation must be assumed for the lachrymal glands. Langley saw a free secretion from these glands after suprarenal injection in the cat, but none in the rabbit. According to Eppinger and Hesz, pilocarpine gives, besides secretion of saliva, also a flow of tears. Emotional secretion of the lachrymal glands is doubtless not a phylogenetically and ontogenetically old reflex, for lower mammals and new-born babes do not shed tears. So the emotions of fight and flight are not accompanied by secretion of tears, as may be observed with persons in rage or in active fear; usually, if weeping occurs, it comes afterwards when relaxation has followed these emotions. In emotions blood-pressure is constantly found increased; now Kornfeld (1899) has found that, whenever the secretion of sweat and of tears began, blood-pressure, which had been increased by the preceding emotions, began to fall.

Sweat glands.—According to Gaskell, sweat glands are only supplied by nerve-fibres from the thoracico-lumbar outflow (sympathetic). Langley did not observe any secretion of sweat after suprarenal injections; Elliot, however, observed that the human hand readily answered by secretion of sweat on an intravenous injection of adrenalin. There is, however, much evidence that here also the glands have a splanchnic innervation (probably from the bulbar

[1] [47], p. 198.

outflow), as the secretion of sweat is much under control of vagotropic drugs (Eppinger and Hesz, "Die Vagotonie": according to them, adrenalin may even stop the secretion). Dupuy (quoted from Higier) has stated that the sweat-glands continue to secrete after the sympathetic nerve has been cut. So Higier (1917) believes it most probable that the sweat glands also have a double innervation. As is well known, all sorts of emotions (perhaps every psychical process, as may be concluded from the so-called psycho-galvanic reflex-phenomenon) may give an increase of sweat secretion. Of the emotions, which we are discussing here, perspiration is most obvious in extreme anxiety, whereas it is much less striking in rage (see also Kornfeld's observations, mentioned above). The utility of sweating is explained by Cannon in the following way (p. 203): "The evaporation of sweat helps to keep the body temperature from rising unduly from the heat of exertion. Again in strong emotion and in pain the 'cold sweat' that appears on the skin may be regarded as a reaction anticipatory of the strenuous movements that are likely to ensue."

Genital organs.—One group of organs remains, which is, *omnium consensu*, innervated by the thoracico-lumbar outflow—i.e., the internal and partly also the external genitals. Gaskell argues that this group also does not form an exception to the rule, that the sympathetic nerves innervate only external regions; for these muscles are in part underlying the skin, in part derived from muscles surrounding the Wolffian and Müllerian ducts; now these ducts are epiblast formations and not derived from the mesoblast. The internal genital organs are supplied by sympathetic fibres only; there seems to be no trace of an innervation from the sacral outflow. The external genitals, however, are reciprocally innervated by the sympathetic system and by the pelvic nerve of the sacral division (Langley, Gaskell). Cannon (op. cit., p. 270) draws attention to the fact that the emotions which appear in these two antagonistic divisions are strikingly opposite; for fear, anxiety, and rage prevent the sexual act or cause inhibition of it in animals and man. He considers the nervi erigentes as the part of the sacral autonomic in which the peculiar excitement of sex is expressed, whereas the sympathetic division is "operated characteristically in the major emotions." This assumption would at the same time explain the opposition of emotional states such as fear and anxiety to the sexual emotions. To me, however, it seems still questionable whether we may consider the nervi erigentes as the principal or even the only route to expression of the sexual emotions. For, as Cannon

himself says (op. cit., p. 271): "When the acme of excitement is approaching it is probable that the sympathetic division is also called into activity; indeed, the completion of the process, the contractions of the seminal vesicles and the prostate and the subsidence of engorged tissues, all innervated by sympathetic filaments, may be due to the overwhelming of sacral by sympathetic discharges." But, though "the sexual orgasm involves a high degree of emotional excitement, it can be rightly considered as essentially a reflex mechanism" (op. cit., p. 33). Erection, however, may often be considered as a reflex, whereas at other times it may occur automatically, without any emotion, e.g., in the morning. Ejaculation and orgasm, on the contrary, though they usually are doubtless reflexes, may sometimes take place without antecedent peripheral stimuli, but may still be accompanied by emotional reactions (pollutions). These objections, however, do not alter that other fact, mentioned by Cannon, that fear or anxiety cause inhibition of the sexual act. And it is certainly a useful thing for the animal in danger, that all energy is drawn away from the vegetative and generative functions and placed into the sympathetic (animal) system, in order to enable the individual to escape from danger.

Sphincter functions.—This brings me to the last question of this chapter. As has been mentioned above, the sympathetic system has an inhibitory influence on the small and large intestines and on the bladder, whereas it supplies with motor fibres the various sphincters (see Gaskell, op. cit., p. 43). Now, as Cannon also points out (op. cit., p. 272), the contraction of the bladder and the rectum in very intense periods of emotional stress, when the sympathetic division is strongly innervated, seems to be a contradiction to the theory advanced in the former pages. Cannon suggests that in such conditions the orderliness of the central arrangements may be upset, but this is no explanation. I might suggest that it is due to the paralysing power of sudden and extreme terror. All striated musculature may then be relaxed; the breathing is interrupted and the heart fails to act. These symptoms are all opposed to those commonly seen in rage and fear, when the sympathetic and the whole voluntary nervous system come into action. In South Africa it is a well-known fact that baboons, when they suddenly encounter a serpent, become totally paralysed; they are unable to flee, and void their excretions (see also Darwin, op. cit., pp. 146 and 292). It is also chiefly at the occasion of sudden terror that we observe the paralysis of the sphincter action in man. This view would be in harmony with Cannon's theory of the energizing power

of emotions by operating the sympathetic division of the involuntary nervous system. For if activating emotions such as rage and fear mean a stimulation to all animal functions it is easily understood that a stronger emotion, such as sudden terror, may paralyse them; it would only be an example of the well-founded biological rule, that a stimulus may cause paralysis on becoming stronger.

Tonus of Striated Musculature.—Finally, I want to mention, for the sake of completeness, the influence which the sympathetic system might possibly have on the tone of the skeletal muscles. It was shown in 1913 by de Boer that resection of the rami communicantes produced a loss of tone in the striated musculature of the frog; these results could also be obtained with cats. The experiments of de Boer were confirmed by Mansfeld, Lukács and Ernst (1915), who examined the chemical tone of striated muscles. Dusser de Barenne (1917) and von Brücke (1918), however, hold the opinion that the influence of the thoracico-lumbar outflow of the involuntary nervous system (sympathetic) on the muscular tone is of small importance only. In studying the literature on this subject one gets the impression that the sympathetic nerve has indeed some influence on the tonus of striped muscles, but that there are also other influences which regulate the muscular tone independent of the sympathetic system. As this question, however, is still undecided and as we have no exact observations, as far as I know, on the muscular tone in mental disorders (which might serve as a comparison) I shall not enter into a deeper discussion of the subject.

Chapter III.—The Sympathetic System in Melancholia.

So the question remains to be answered, whether in psychiatry there are more signs of sympathetic stimulation besides the increase of blood-sugar (see p. 254). As I have made a closer study of the blood-sugar in melancholia, and as we also have the most reliable information about bodily changes in this disease, I have chosen melancholia for testing this question. I want to declare emphatically that I make no claim to be complete; there may also be other psychoses with symptoms of increased sympathetic innervation, e.g., amentia; I have only taken melancholia as an example.

Intestinal Disorders.

Under this title I shall also discuss the *salivary glands* and for convenience' sake together with them the *sweat glands* and the *lachrymal glands*. Whether these glands are supplied with secretory fibres chiefly by the splanchnic or by the sympathetic division, or finally by both systems, is a question which I have discussed on pp. 259-260.

In the emotional states which cause an increase of sympathetic activity, we certainly find inhibition of salivary secretion, there is no sign of lachrymal secretion, and sweating is a process which is combined rather with the paralysing than with the activating kinds of emotions which operate the sympathetic system. All these secretions are often diminished in melancholia; by most authors this fact is considered to be an example of the general inhibition. The inhibitory influence of the disease would not only appear in the well-known motor inhibition, but also in the decrease of secretions, of the intestinal movements and of the pulse. That this conception is wrong may be directly deduced from the fact that secretions and intestinal movements are diminished most in cases of anxiety, in which there is no question of a general inhibition. That salivary secretion is often diminished in melancholia was observed by several psychiatrists (e.g., Ballet, 1903, Ziehen, 1908, Church and Peterson, 1914), though a stuporous melancholic may give the impression of increased salivation by retaining the saliva in his mouth for a long time. It is, however, quite different from the true salivation in a catatonic stupor. Of course this is a difficult subject for exact examination.

We find in nearly all text-books the observation that weeping is very seldom seen with melancholia. Also in the anxious form, *lachrymal secretion* is usually totally absent, patients even complain of being unable to weep (Ziehen, Kraepelin, 1913, Stransky, 1911). The *secretion of sweat* and its relations to the sympathetic division and the discussed emotions are a problem of some difficulty, as is explained on p. 260. Most authors speak of a dry, scaly skin in melancholia (Ziehen, Kraepelin, Stransky), Bruce and de Maine Alexander (1900) mention a dry skin in the acute form (amentia?).

Gastric Secretion.

Through the fundamental experiments of Pawlow (1898) we know the activating influence of special sensory and psychical stimuli of an agreeable kind on the salivary and gastric secretion. This fact also holds good for man, as was proved by A. Bickel (1906), Cade and

Latarget (1905), and Herz and Sterling (experiments with gastric fistulæ in man). On the contrary, it appears that emotions, such as worry, fear and anger, have a strong inhibitory influence on the secretion of gastric juice in the dog (A. Bickel and Sasaki, 1905), and even on the secretion of pancreatic juice and bile (Oechsler, 1914, quoted after Cannon, op. cit., p. 13). It is also a fact that the sensory and psychical stimuli which cause secretion of gastric juice are conducted by the vagus nerve (Pawlow); whether it is the splanchnics that conduct the inhibitory stimuli in fear and anger is still undecided. Most examinations we possess on the gastric secretion in melancholia are of older date. The first to study the question was von Noorden (1887), who concluded that in melancholia the amount of HCl is increased, that the emptying of the stomach is accelerated, and the digestive power of the juice is normal. Most of von Noorden's cases, however, were not suffering from maniacal-depressive insanity or from simple melancholia, but from dementia præcox, confusional insanity, and other psychoses. So, although these examinations were carefully performed, we cannot use them for our present problem. And this is the case with most other publications of older date. Of the eleven observations by Pachoud (1888), there are only four (Observation V, VI, VII and XI) which, according to our present conception, might be cases of melancholia. Of Orliac's (1892) six cases only the first belongs to this form of insanity; this one showed a low amount of HCl. Pachoud fully agrees with von Noorden. Placzek (1890), on the contrary, who examined eight cases of melancholia (all probably true examples of the disease, except No. 8), found a very low percentage of HCl and a slow emptying of the stomach. The results of Leubuscher and Ziehen (1891) varied, the cases of melancholia showing hypo- and hyper-acidity and also normal conditions. In the same way Bell (1909) found partly subnormal, partly hypernormal amounts of HCl in melancholia. Cowie and Inch (1905) observed in 81·8 per cent. of their twenty-two cases hyperacidity; they found the digestive power of the gastric juice increased, though it is not secreted in large quantity. E. Mayr (1907) and Togami (1913) also paid attention to the secretion of pepsin and rennin. Mayr does not speak of melancholia specially, but he combines all psychogenous mental disorders, in which anxiety psychoses (also melancholia?) are included. In these he finds a high amount of HCl and a very small amount of pepsin, and also only a little rennet. According to Mayr, these conditions are characteristic of all emotional states. Togami

has obtained about the same results. Of his six cases of melancholia, however (of which the first case, No. 21, surely is not a good example of melancholia), only two (Nos. 23 and 26), show a hyperacidity, whereas Nos. 24 and 25 have a rather low percentage of HCl, and No. 22 a normal amount. The amount of pepsin, however, examined by the edestin reaction, was found to have constantly decreased; indeed, pepsin is often nearly absent. Summing up, we can say that the question of the percentage of HCl is still undecided.

In addition to the fact that various authors have examined different forms of insanity, there are still other reasons for the incongruity of the above-mentioned results. First of all, the meals used by the patients were different, and thus at the same time the chemical and psychical stimuli for gastric secretion. Secondly, we must not only look for a change of the quality of gastric juice, but also for the quantity; for A. Bickel (1906) has shown that the amount of HCl is fairly constant, whereas the quantity is increased or diminished in different psychical states. The amount of pepsin, however, is almost constantly found to be decreased. At any rate we may take it for granted that emotions such as worry, fear and anger have a strong inhibitory influence on gastric secretion, not only in animals, but also in normal persons. The instances of Hornborg (1904), of Bogen (1907), and of Herz and Sterling, all experimenting on gastric fistulæ in man, have put the question beyond doubt; Grandauer's researches (1911) are a valuable confirmation of these experiments.

Motility of the Intestine.

The same can be said of the influence of worry, fear and anger on the motility of the stomach and of the small and large intestines. This too we owe to the researches of Cannon and his school. Cannon found in 1898 that these emotions caused inhibition of the peristalsis of the stomach; by means of Röntgen-rays he proved in 1902 that the same was the case with the small and large intestines. "Like the peristaltic waves in the stomach, the peristalsis and the kneading movements of the small intestine all cease whenever the observed animal shows signs of emotional excitement. (Cannon, 1918, op. cit., p. 16.) These observations are not only true for the cat, but also for the dog, the rabbit and the guinea-pig; they are confirmed by other authors (Lommel, 1903; Auer, 1907; Katsch, 1913). On the contrary, the emotion of seeing the desirable food is, as with gastric secretion, sufficient to cause peristalsis of the

stomach (Best and Cohnheim, 1910) and of the intestine (Katsch, 1913). Now the vagus nerve supplies with motor fibres the stomach and the small and large intestines (see also the experiments of Cannon, 1906); the splanchnic (sympathetic division) sends inhibitory fibres to the same muscles and motor fibres to the various sphincters (Gaskell). Accordingly the injection of adrenalin causes a sudden stoppage of the movements and at the same time anæmia of the whole intestine (Katsch, 1913). As the vagus is without doubt the motor nerve for the stomach and the small intestine, it is almost certain that the psychical stimuli which cause peristalsis[1] are conducted by this nerve; the direct proof, however, is not yet given, as far as I know. That it is the sympathetic system which conducts the inhibitory stimuli dependent on fear, anxiety, &c., is very probable. Katsch saw through his "experimental abdominal window" when the animal (rabbit) was startled, e.g., by the sudden fall of a heavy object, that the intestine suddenly became quite pale and absolutely motionless; it is, as he writes, "as if we had made an intravenous injection of adrenalin; in quite the same way the inhibitory influence of terror acts by means of splanchnic stimulation." Indeed, the paleness accompanying the motor inhibition makes it certain that it is the splanchnic nerve which conducts the inhibitory stimuli of emotion. Katsch saw the same effect from pain, in accordance with Cannon's researches, who found the same reactions of sympathetic stimulation and of increase of adrenalin-secretion in pain and in fear and anger. Hotz (1909) found that the inhibitory influence of disagreeable sensory stimuli is bound up with the splanchnic, for it is absent after splanchnic section. The brief remarks of Fubini (1892) and Esselmont (1899), who observed an activating influence of emotions such as fear on the intestinal motility, in my opinion cannot upset the well-founded and often confirmed conception of Cannon.

Whereas it is certain that the mechanism of gastric secretion is the same in animals and man, we have but little proof of the inhibitory influence of emotion on intestinal motility in normal man. Rosbach (1890), however, observed in a woman with an anterior colotomy and with a remarkably thin abdominal wall (so that he was able to study the influence of various stimuli on peristalsis in the same way as Katsch

[1] The word peristalsis is not used in the sense of a contraction of the gut above a food mass and a relaxation below it, in contradistinction to the other intestinal movements, such as pendular and rolling movements, but in the general sense of movements of the intestinal tract.

No.	Sex	Age	Name	Constipation	Prescriptions	Remarks on psychical state
1	F.	43	S. D. B. ..	2·3	No purgatives, 3 × 40 mg. opium p. d.	Slight case, no anxiety, bed ; P.
2	F.	72	Mrs. S. ..	2·1	4 × lax., no opium ..	Slight case, no anxiety, bed ; P.
3	F.	52	Mrs. Sch. ..	2·3	No lax., no opium ..	Marked anxiety, walking ; P.
4	M.	78	P. P. ..	3·5	6 × lax., ± 13 mg. opium p. d.	Marked anxiety, bed ; P.
5	F.	36	Mrs. Sc. ..	1·3	No lax., no opium ..	No anxiety, bed ; P.
6	F.	72	Mrs. B. ..	2·0	No lax., no opium ..	No anxiety, bed ; P.
7	M.	18	D. R. ..	1·5	No lax., no opium ..	Stupor, bed ; I.
8	F.	60	Mrs. H. ..	1·2	No lax., no opium ..	No anxiety, bed ; I.
9	M.	48	H. F. ..	1·1	No lax., 3 × 40 mg. opium p. d.	No anxiety, bed ; P.
10	M.	50	H. N. ..	1·2	1 × lax., no opium ..	No anxiety, bed ; I.
11	F.	30	Mrs. V. ..	1·5	No lax., no opium ..	Slight case, no anxiety, bed ; I.
12	M.	48	H. B. ..	2·7	8 × lax., no opium ..	Sometimes marked anxiety, bed ; I. ; otherwise motor inhibition
13	M.	26	J. H. ..	1·5	No lax., no opium ..	Marked anxiety, bed ; P.
14	F.	40	Mrs. M. ..	1·1	No lax., no opium ..	Slight case, no anxiety ; I.
15	F.	55	K. S. ...	1·3	No lax., 3 × 60 mg. opium p. d.	No anxiety; bed ; I.
16	F.	63	Miss P. ..	1·1	No lax., no opium ..	No anxiety, walking ; P.
17	M.	43	J. v. R. ..	1·5	No lax., no opium ..	No anxiety ; I.
18	M.	52	F. F. ..	1·0	No lax., no opium ..	No anxiety, bed ; I.
19	F.	35	G. K. ..	1·2	No lax., ± 15 mg. opium p. d.	No anxiety ; I.
20	M.	55	W. v. D. ..	2·1	6 × purg. p. m., + 2 × lax. p. d., 5 × 50 mg. opium p.d.	Permanently slight degree of anxiety, bed ; I.
21	M.	61	W. S. ..	2·7	5 × purg. p. m., + 1 × lax. p. d., no opium	Marked anxiety, could scarcely stay in bed ; P.
22	M.	46	D. A. ..	3·2	9 × purg. p. m., + 1 × lax. p. d., 3 × 60 mg. opium p. d.	Very anxious, could scarcely stay in bed ; P.
23	F.	50	A. G. S. ..	4·7	5 × lax. p. m., 3 × 60 mg. opium p. d.	Anxiety + motor inhibition, glycosuria, bed ; P.
24	F.	44	H. L. ..	2·0	6 × lax. p. m., no opium	Stupor, no anxiety, partly in bed, partly walking about ; P.
25	M.	26	O. P. ..	2·0	1 × lax., no opium ..	Not anxious, bed ; P.
26	M.	43	B. D. ..	1·6	No lax., no opium ..	Not anxious, bed ; P.
27	M.	65	H. B. ..	2·3	1 × lax., no opium ..	Anxiety, glycosuria ; I. disappearing on recovery, bed
28	F.	48	M. M. ..	2·0	1 × lax., 2 × 70 mg. opium p. d.	Slight degree of anxiety, partly in bed, partly walking about ; P.
29	F.	47	M. D. O. ..	3·4	3 × lax., 4 × 60 mg. opium p. d.	Very anxious, walking about ; P.
30	F.	50	H. F. H. ..	1·2	No lax., no opium ..	No anxiety, bed and walking ; P.
31	F.	29	R. P. de V. ..	2·4	1 × lax., no opium ..	Slight degree of anxiety. bed ; P.
32	F.	56	H. B. B. ..	2·8	7 × lax., no opium ..	Marked anxiety, bed ; P.
33	F.	34	D. O. D. ..	1·3	No lax., no opium ..	Soon convalescent, no anxiety, partly in bed, partly walking about ; P.

No.	Sex	Age	Name	Constipation	Prescriptions	Remarks on psychical state
34	F.	61	L. S. B. ..	2·8	2 × lax., 4 × 50 mg. opium	Anxiety, bed ; P.
35	F.	45	A. K. H. ..	1·4	No lax., no opium ..	Not anxious, bed and walking ; I.
36	M.	45	H. Ph. R. ..	2·5	8 × lax., no opium ..	Anxiety and motor inhibition, bed ; P.
			Same patient	1·0	Half glass of apenta p. d., no opium	No longer anxious, motor inhibition, bed
37	F.	30	R. de V. ..	1·9	No lax., no opium ..	Not anxious, bed ; P.
38	M.	57	K. R. ..	1·4	2 × lax., no opium ..	Not anxious, bed, walking ; P.
39	F.	40	A. G. ..	2·0	2 × lax., no opium ..	Not anxious, walking about ; P.
40	F.	51	G.v. d.V.v. D.	2·8	1 × lax., no opium ..	Anxiety, bed ; P.
41	F.	41	B. M. ..	2·2	6 × lax., 4 × 40 mg. opium p. d.	Not anxious, bed, walking ; P.
42	M.	50	K. V. ..	1·3	No lax., no opium ..	Not anxious, bed ; P.
43	F.	54	G. D. J. ..	1·3	No lax., no opium ..	Not anxious, a single time excepted, bed ; P.
44	F.	51	K. M. v. d. K.	1·4	No lax., no opium ..	Not anxious, bed ; P.
45	M.	38	A. v. C. ..	1·0	No lax., no opium ..	Not anxious, bed ; I.
46	F.	45	W. M. L. ..	1·5	No lax., no opium ..	Not anxious, bed, walking ; P.
47	F.	33	J. B. v. d. W.	2·8	8 × lax., 3 × 30 mg. opium p. d.	Anxiety, could scarcely stay in bed ; P.
48	F.	29	G. R. ..	1·5	No lax., no opium ..	Not anxious, bed, walking ; I.
49	F.	52	E. B. v. d. P.	2·3	1 × mild lax. p. d., 3 × 40 mg. opium p. d.	Anxiety, could scarcely stay in bed ; P.
50	F.	44	H. S. S. ..	1·6	5 × lax., no opium ..	Stupor, no anxiety, bed ; P.
51	F.	59	H. N. S. ..	1·7	2 × lax., no opium ..	No anxiety, bed ; P.
52	F.	52	H. W. G. ..	2·4	6 × purg. p. m. + 1 × lax. p. d., no opium	Anxiety, bed ; P.
53	F.	50	C. K. B. ..	3·7	9 × lax., 3 × 60 mg. opium p. d.	Anxiety, bed ; P.
54	M.	58	B. J. D. ..	2·0	No lax., no opium ..	No anxiety, bed ; P.
55	F.	41	B. F. de L. ..	3·1	6 × lax., 3 × 50 mg. opium p. d.	Anxiety, bed ; I.
56	F.	41	A. H. ..	1·9	1 × lax., 3 × 50 mg. opium	Slight degree of anxiety, mostly calm, bed ; I.
57	M.	45	D. L. ..	3·3	7 × lax., no opium ..	Anxiety, could scarcely stay in bed ; P.
58	M.	47	W. W. ..	1·4	No lax., no opium ..	Not anxious, bed ; P.
59	M.	44	E. de H. ..	4·0	4 × lax., no opium ..	Anxiety, bed ; P.
60	M.	43	H. L. ..	1·2	No lax., no opium ..	Not anxious, bed, walking ; I.

P = Periodical melancholia.
I = Melancholia for the first time, but recovered on leaving the hospital.
Purg. = stronger purgative, such as oleum ricini, cascara, clysma, &c.
lax. = mild purgative, such as saline waters, which may be given to the patient every day.
p. m. = during four weeks.
p. d. = pro die.

could by means of his "Bauchfenster") that with emotions such as fear and worry all movements of the intestinal tract suddenly ceased; this inhibition lasted for five till ten minutes. The same emotion never caused peristalsis to start in the formerly quiescent bowel.

What do we know of this influence in mental disorders, especially in melancholia? Of the motor power of the stomach in melancholia we have little information. Bruce and de Maine Alexander observed a weak motor power in acute melancholia, but probably their cases were amentia. Placzek found the same in cases of presumably true melancholia, whereas van Noorden and Pachoud found the emptying of the stomach accelerated. I have already criticized these publications on p. 265. With the knowledge of the general effect of adrenalin or stimulation of the sympathetic and of emotions such as fear and fright, both on the motility of stomach and the intestines, and of the weak motor power of the small and large intestines in melancholia, it seems probable that the motor power of the stomach is also inhibited in this disease. The researches of Holzknecht (1911), who found by means of the Röntgen rays a decrease of tone of the œsophageal wall in cases of "Cyclothymie" (mild form of maniacal-depressive insanity), also point in this direction.

The inhibition of the intestinal motility is so widely known and so generally admitted, that I need not mention any literature on the subject. One can find it described in every text-book as a symptom characteristic of melancholia. It is generally attributed to the typical motor inhibition and general want of movements of the patient. With our knowledge of the above-mentioned physiological facts, it is superfluous to emphasize the improbability of this explanation. But the clinical data are also unfavourable to this conception. Firstly, we may find the symptom in mild forms of the maniacal-depressive insanity (cyclothymia, Willmanns, 1906, Roemheld, 1908), in which we do not find any motor inhibition of importance, but chiefly depression, and at the same time agitation and restlessness. But then we may find obstinate constipation in cases of anxious melancholia, though these sufferers move restlessly all day long. I have examined the morbid histories of sixty cases of melancholia I observed; I have only chosen periodical cases, and, of patients who had their first attack only, those who had recovered completely on leaving the hospital. I must emphatically state that I have chosen cases of true melancholia only; none of them suffered from dementia, from confusion or hallucinations or from symptoms which might suggest dementia præcox; all bodily

changes were absent. With these precautions, only cases of genuine melancholia remained, and I deemed it permitted to give the results in the preceding table, without giving the clinical reports. It is always noted on the charts of our hospital when a patient's bowels were opened, and when he received a purgative or any other medicine. As I also disposed of the reports on the psychical state of the patient, I was able to put together the following table. I have taken the number of days during which the patient was observed to be fully melancholic (at least during four weeks) and I have divided this number by the number of motions during that time. The resulting numbers are noted down in the table under the title: Constipation. This, however, would give a false impression of the intestinal motor power, since purgatives are usually prescribed for patients suffering from constipation; many of them had hardly any defæcation at all without laxatives. On the other hand opium is often prescribed in anxious melancholia, and this medicament may have an inhibitory influence on intestinal motility. So I noted down under the title "Prescriptions" how often the patient was given a purgative during the four weeks, and how much opium he had taken daily. In the last column the reader will find remarks on the psychical state of the patients, and whether they were bedridden or were allowed to walk about. The description, "not anxious," of course does not mean that there was no sign of anxiety during the whole disease, but only that depression and motor inhibition were the predominant symptoms, and that anxiety occurred only slightly or occasionally.

As may be seen from the table, the cases with marked anxiety not only show constipation like the not-anxious cases, but they do so in a much higher degree. Nevertheless, in these cases anxiety had broken the motor inhibition, the patients moved restlessly about and could scarcely be kept in their beds. When we single out the cases in which permanent anxiety was the most striking symptom (Nos. 3, 4, 13, 21, 22, 23, 27, 29, 32, 34, 36, 40, 47, 49, 52, 53, 55, 57, 59), we get the following numbers:—

Average of constipation	..	3·0
Average of purgatives	..	4·6 (+ in 20 per cent. of the cases a mild laxative once a day)
Average of opium	..	77 mg. pro die.

Whereas for the remaining "not anxious" patients[1] these numbers are:—

[1] In this number the cases are included which showed a slight degree of anxiety or which presented the symptom occasionally only.

Average of constipation	..	1·6
Average of purgatives	..	1·1 (+ in 7 per cent. of the cases a mild laxative once a day)
Average of opium	..	27 mg. pro die.

This means that, though the anxious melancholics used much more purgatives (namely, on an average 4·6 times during four weeks, which is four times more than the "not anxious" patients received), they defæcated only once in three days (to once in 1·6 days in the "not anxious" cases).

Of course, opium was given much more frequently in the "anxious" cases; this, however, cannot explain the difference, for, if we consider only *those cases of anxious melancholia in which no opium was prescribed* (Nos. 3, 13, 21, 27, 32, 36, 40, 52, 57, 59), we find for the *average of constipation* the number 2·7, whereas the *average of purgatives* is still much higher than with "not anxious" patients, namely, 3·9 times a purgative and in 20 per cent. of the cases a mild laxative once a day. Therefore I think it very probable that constipation in melancholia is caused by emotions, analogous to the fact that the same emotions cause motor inhibition of the intestines in animals.

Vascular Changes.

As I have mentioned on p. 217, the secretion of adrenalin was increased in emotions such as anger and fear (Cannon and de la Paz, Elliot); it is also well known that injections of adrenalin cause an increase of blood-pressure and have a stimulating effect on the heart, at least on the isolated heart. It was, therefore, *a priori* highly probable that emotions in the animal would cause an increase of blood-pressure and an accelerated pulse. The latter fact needs no further discussion, it can be observed in every excited animal.

Blood-pressure.—That pain, fear and anger also cause an increase of blood-pressure is considered by Cannon as a fact beyond a doubt, as it is the direct consequence of the increased sympathetic stimulation in emotions: "Great excitement is accompanied by sympathetic innervations which increase the contraction of the small arteries, render unusually forcible the heart beat, and consequently raise arterial pressure (op. cit., p. 95). I do not know, however, whether exact experiments to prove this increasing influence of emotions in animals[1]

[1] Of course it is a well-known fact that stimulation of afferent nerves causes an increase of blood-pressure in animals; the influence of emotions on the blood-pressure in curarized animals was studied by Couty and Charpentier (1877) and by E. Weber (1910); these experiments gave no constant and trustworthy results.

have been made, nor can I find any account in the monograph of Cannon. Still, I take it for granted that such a careful examiner as Cannon, who has observed so many animals in emotional state, has sufficient proof for his positive assertion. That a high blood-pressure might be extremely useful in the muscular activities which are likely to accompany emotions such as rage and fear is pointed out very clearly by Cannon (op. cit., pp. 107 and 200), as I have already mentioned on p. 255. It is well known that emotions cause an accelerated pulse in normal persons; the emotional rise of blood-pressure in man is also proved by many experiments. Of the former authors on the subject, I will only mention Kiesow (1895) and Binet and Vaschide (1897); both investigations were made by means of Mosso's sphygmomanometer. These examiners observed that every psychical process, whether it be a sensorial perception, or thinking, talking, calculating, causes a constant rise of blood-pressure. Most of all, however, the vascular tension is affected by emotions: according to Binet and Vaschide, sudden emotion, both of an agreeable and of a disagreeable kind, causes the greatest degree of increase, as high as is produced by muscular labour. The increasing influence of emotions on blood-pressure is confirmed by numberless examiners, using different sphygmomanometers, e.g., by Geisböck (1904), Ziehen (1908), Haškovec (1905), Heim (1900), Grebner (1900), Kornfeld (1899), Gellhorn and Lewin (1913). Even the mere fact of the blood-pressure being examined for the first time is sufficient to cause a rise of blood-pressure in a patient, and only the later observations give reliable results. It is not surprising, therefore, that in melancholia also blood-pressure is found to be high by nearly all authors. The first publication on the subject was made by Cramer (1892), experimenting with the manometer of von Basch. By these careful examinations it was made clear that blood-pressure is high in melancholia, especially in the anxious form. The investigations of Craig (1898) are apparently in contradiction to Cramer's results. Craig divides the psychoses into two groups: those with excitement with low pressure and those with depression with high pressure; with agitated melancholia he came to varying results. This division is doubtless incorrect if in the word "excitement" the melancholic agitation and anxiety are also included; probably the author's conception of excitement is a maniacal condition. For if we consider his results in melancholia and in mania, it appears that this author also finds in melancholia a high blood-pressure and a low pressure in mania. This is also Broadbent's (1898) opinion: "The

tension is usually high in melancholic conditions and usually low in maniacal conditions." Pilcz (1900), using Gärtner's tonometer, examined 240 patients suffering from mental disorders; according to him, blood-pressure is constantly increased in melancholia, especially in anxiety. In mania the pressure is usually low, though it may be raised in fits of anger; moreover, it has no constant height in mania, but varies frequently. Alexander (1902) also examined blood-pressure in melancholic and maniacal conditions, with the help of Barnard's and Hill's sphygmomanometer. Speaking generally, he found a high pressure in the acute, emotional form of both diseases; perhaps, according to our present conception, many of his cases of acute melancholia belong to the amentia group. Alter (1905) also often observed with Gärtner's tonometer the rise of blood-pressure in melancholia and the decrease in mania, though in the latter it may also be increased. Ziehen (1908) mentions a high pressure in melancholia, especially in the anxious form. Morgenthaler (1910), who used Riva-Rocci's manometer, stated that blood-pressure need not be increased in simple melancholia, but it generally is in the excited form. We owe very careful investigations to Weber (1910), who examined the diastolic and the systolic pressure with the help of Riva-Rocci's manometer (corrected by von Recklinghausen). Weber found, first, a rather normal pressure in quiet melancholia; second, supranormal numbers in excited melancholia, the systolic pressure especially was increased; when the patients become calmer the pressure falls; third, in stupor the pressure is usually increased; fourth, in quiet the blood-pressure is normal, whereas, fifth, it is usually increased in cases of excited mania.

I wish to mention the examinations of Longworth (1911) who used the instruments of Riva-Rocci and of Hill and also found a much higher pressure in melancholia than in mania, but who attributed this difference to the fact that the patients suffering from mania were much younger than the melancholics (on an average 48·5 to 33).

Summing up, we may state that blood-pressure is increased in melancholia and most constantly in the anxious form. It is the emotional state that is responsible for the rise of blood-pressure, analogous to the physiological effect of the above-mentioned emotions. That blood-pressure is also found increased in stupor does not speak against this conception; for melancholic stupor is usually an emotional state, as will be discussed at the end of this paper. So it is not surprising that we also find the rise of blood-pressure in other psychoses,

when the patients are in an emotional state. Haškovec (1905), e.g., found it increased in the emotional form of neurasthenia; Heim (1900) in neuropathic children, and Alter in emotional cases of dementia præcox. The results with mania are much less constant than those with melancholia, as may be seen from the above account. It is the same problem as I have discussed in the first chapter, when speaking of blood-sugar in mania. First there are the same difficulties with the examination itself. Cases of real mania are rather rare and as even small muscular labour causes a marked increase of blood-pressure, the results in mania cannot be very reliable. At any rate it is impossible to examine maniacal patients regularly at the same time every day, as they are often restless to such a degree as to make all investigation impossible. But even if we do not consider this bodily reason, there remain the mental causes for a varying blood-pressure; for the maniacal patient is now calm and optimistic and then again restive, furious and highly excited. This must be the reason that the results of the various authors as regards the blood-pressure in mania differ so greatly. Just as blood-sugar is increased in the highly emotional form of mania, so also blood-pressure is raised in the same conditions.

Plethysmographic and Sphygmographic investigations.—As we have seen, injections of adrenalin or stimulation of the sympathetic are followed by a rise of blood-pressure; we know that in emotional states the sympathetic system is over-excited and we also know that the same emotions cause an increase of blood-pressure. It is easy to see in these facts a causal connexion.[1] A direct proof, however, is wanting. I have looked for this proof in the plethysmographic investigations, for the latter were made after adrenalin had been injected, and in the case of emotions with normal persons, and in psychoses. According to Cannon (1918, op. cit., p. 200), the studies of volume changes in parts of the body, made by Oliver and Schäfer, showed that injected adrenalin drove the blood from the abdominal viscera into the central nervous system, the lungs, the heart, and the active skeletal muscles. Oliver and Schäfer (1895) themselves are a little more reserved: "It may fairly be assumed that although we are unable to record any plethysmographic observations upon the intestines, the great rise of blood-pressure, which invariably follows the injection of the extract, is in all cases due very largely to the contraction of the arterioles of

[1] Pain, which has the same sympathetic reactions as anger and fear—as pointed out by Cannon—also gives a marked increase of blood-pressure (see, e.g., H. Strauss, 1901).

the splanchnic area" (op. cit., p. 246). Strong evidence in favour of the opinion that the emotional rise of blood-pressure in animals is really caused by increased adrenalin-secretion, is given by the experiments of von Katsch (1913) who, with the help of his experimental abdominal window observed a sudden pallor and stoppage of the intestines, as well after injection of adrenalin as after sudden emotions.

With man we have many plethysmographic investigations. E. Weber (1910), and after him H. Bickel (1916), studied the changes of volume of the intestines, the cerebrum and the extremities in man during various psychical processes. According to them, there would be a certain antagonism between the external parts of the body and the intestines; the volume of the arm and of the ear would decrease and that of the bowels would increase in the case of intellectual work, or when the attention is fixed upon a sensory stimulus or if the person is unpleasantly struck by the sensory stimulus ("sensorielle Aufmerksamkeit" and "sensorielle Unlust"). On the contrary, the volume of the arm would increase and the volume of the intestines would decrease in the case of pleasant feelings (Weber). These facts are apparently contradictory to our conception given above. That the volume of the arm shrinks in emotions would be no obstacle, for the smaller arteries are contracted by adrenalin; Oliver and Schäfer found as the usual result of their injections a decrease of the volume of the limbs. But the increase of the intestinal volume is a difficult point.[1] One might suppose that strong emotions, such as fear and anger, have other bodily reactions than the slight emotions caused by sensory stimuli or intellectual work. Weber, however, found the same increase of the intestinal volume in sudden terror. Yet I do not think these results to be sufficient proof against our theory; the striking constriction of the intestinal vessels in emotions, directly seen by Katsch, is too much in favour of it. Weber's method of studying the intestinal vascular volume demands many precautions. Professor Wiersma told me that he could not obtain any reliable results by the same method. I doubt whether the author has sufficiently considered the influence of emotions on the intestinal musculature, either of the wall or of the sphincter ani. As Weber brings a balloon of rubber into the rectum (the balloon is

[1] In muscular labour, even if only the intention to move exists (Weber, 1913), we see a general widening of the muscular vessels and constriction of the intestinal vessels. It needs no explanation that this is very useful for man and animals in the way pointed out by Cannon.

then blown up and is connected with a kymograph by a little tube), it is clear that contraction, relaxation and tone of rectum and anal sphincter may influence the pressure of the air in the balloon and cause their special changes in the plethysmogram.

The sphygmogram is no more capable of solving the problem. Wiersma[1] (1911-13) found that the irregularities, which are normally caused in the sphygmogram and in the plethysmogram by respiration, disappear in preoccupation and emotions; the same is found in mania and in melancholia, especially in anxious melancholia. As we do not know whether the same occurs in sympathetic over-excitement, and as therefore a comparison is impossible, these investigations cannot help us to solve our problem. But it shows once more that it is the emotional side which causes the bodily changes in melancholia, not the inhibition; in stupor the curve shows the same regularity, even then the melancholic is highly emotional; this also explains the high blood-pressure and the increase of sugar in the blood in melancholic stupor. We often had the occasion to note the similarity of the bodily changes in mania and in melancholia, both dependent on an increased emotional state. These investigations by Wiersma confirm this conception. Ziehen also (1908) had observed that the plethysgmographic curves in mania and in melancholia are much like each other, if the maniacal excitement is well pronounced (op. cit., p. 601). Summing up, we may therefore state that the rise of blood-pressure is very probably due to an over-excitement of the sympathetic system, but that the absolute proof is wanting.

The question of the *pulse-rate* still remains to be discussed. Though it is the usual text-book statement that it is decreased in melancholia, in this general sense it is doubtless incorrect We may find a slow pulse in melancholia, even in the anxious form, but more often it is accelerated. P. Weber (1910) showed that on an average the pulse-rate is increased both in melancholia and in mania. These facts are in perfect harmony with our conception about over-excitement of the sympathetic division in melancholia. For adrenalin causes an increase of rate of the isolated heart; if, however, adrenalin is brought into

[1] These results are confirmed by Küppers (1913). Ziehen, Saiz (1907) and H. Bickel, on the contrary, observed very pronounced respiratory oscillations in emotions and in the maniacal depressive psychoses. The large number of curves made in many normal and pathological cases by Wiersma, all showing a regular pulse in emotions, have convinced me that this fact is beyond a doubt. Moreover, they have been confirmed by the simultaneous registration of the psycho-galvanic reaction (Wiersma, 1915).

the circulation of a normal animal, it causes at the same time constriction of the smaller arteries and consequently a rise of blood-pressure, which has a depressive influence on the heart-rate. Hoskins and Lovelette have proved (1914) that in the same animal injection of adrenalin may cause an accelerated pulse, if the blood-pressure had not risen too much, and a decrease of the pulse-rate if the blood-pressure had become very high. In most of their cases, however, they observed an increase of the pulse-rate together with an increase of blood-pressure.

Reflex Dilatation of the Pupil.

All psychical processes cause a widening of the pupil; anxiety may do so in a high degree. It is a well-known fact that anxious persons have large pupils, and all mental diseases in which anxiety occurs show this phenomenon. Observations of this kind in melancholia can only be found scattered about in literature; Bumke mentions a case of anxious melancholia in which the pupil was every now and then dilated to such a degree that even the light reflex could no longer be obtained. The only systematic investigation of the size and reactions of the pupil in melancholia known to me is that by Weiler, who examined principally stuporous states of maniacal-depressive psychosis. He never found miosis, but, on the contrary, often mydriasis. The psychical reflexes, even in complete stupor, were never absent; but in 10 per cent. the reactions were not well pronounced, and these were the doubtful cases with paranoid symptoms; in the cases of typical melancholic stupor, however, the psychical reactions were always very easily and clearly obtained. This is again a marked difference from catatonic stupor. Van der Scheer (1915, op. cit., p. 469) found that the adrenalin-mydriasis is frequent in maniacal-depressive insanity; the number of cases examined, however, was small. Mydriasis after instillation of adrenalin in the eye is a rather intricate reaction. A. Loewy and Rosenberg (1914) found that the so-called "O. Loewisches Pupillenphänomen" (i.e., adrenalin-mydriasis in the dog after extirpation of the pancreas) was present in all cases of experimental hyperglycæmia; in this way the phenomenon would not have the meaning of a specific reaction on sympathetic over-excitement. Pötzl, Eppinger and Hesz (1910) have tested O. Loewi's phenomenon in different forms of insanity; their results with the maniacal-depressive psychoses were varying; in some cases the reaction was strikingly positive, in other cases it was negative.

Conclusions.

We have found in melancholia many signs of over-excitement of the sympathetic system, or of hypersecretion of adrenalin. In many points this problem is still undecided, and we often saw reason to conclude that the final proof was wanting. I do not therefore put forward this conception as a solidly based theory, but as a working hypothesis only.

I am aware of the fact that I have not solved the problem in all its details; but I believe that the principal bodily changes in melancholia can better be understood now that they have been observed from one point of view. For I think it certain that they are all dependent on the highly emotional character of the disease, not only the increased blood-sugar, but the intestinal disorders, the rise of blood-pressure, and the dilatation of the pupils. In this respect it does not differ so much from mania as has often been supposed, at least not from the fully excited form. For in both conditions, in melancholia and in mania, the increased emotionalism is the principal point. We again find it in the character of the patients, even in their normal state, and in the character of many of their relatives. Emotionalism is the most constant symptom of both diseases, for it is found in stupor of melancholia as well as in its restlessness and anxiety; we find it in mania and also in the mixed form of the disease, which forms the connecting link between the two apparently so different phases. Emotionalism makes melancholia and mania an undeniable entity: the maniacal-depressive psychosis and the various bodily reactions, discussed in this paper, are its direct consequences.

It is not surprising that these reactions are most constant in melancholia. For the emotions of a depressive character are, phylogenetically spoken, old, and they have been provided with the bodily reactions which are useful to the animal in attack, defence or flight. I may here draw attention to the remarkable fact that the depressive emotions of the melancholic and the excitement of the maniacal are essentially different from the grief and the joy of normal persons. For melancholic depression is always mixed with a certain agitation and anxiety, maniacal excitement with a certain irritability, a tendency to resist or to fight.

The attacks of the maniacal-depressive psychosis overtake their victims without our being able to perceive any real corporeal or psychical reason. Would not phylogenesis help us to find the deeper

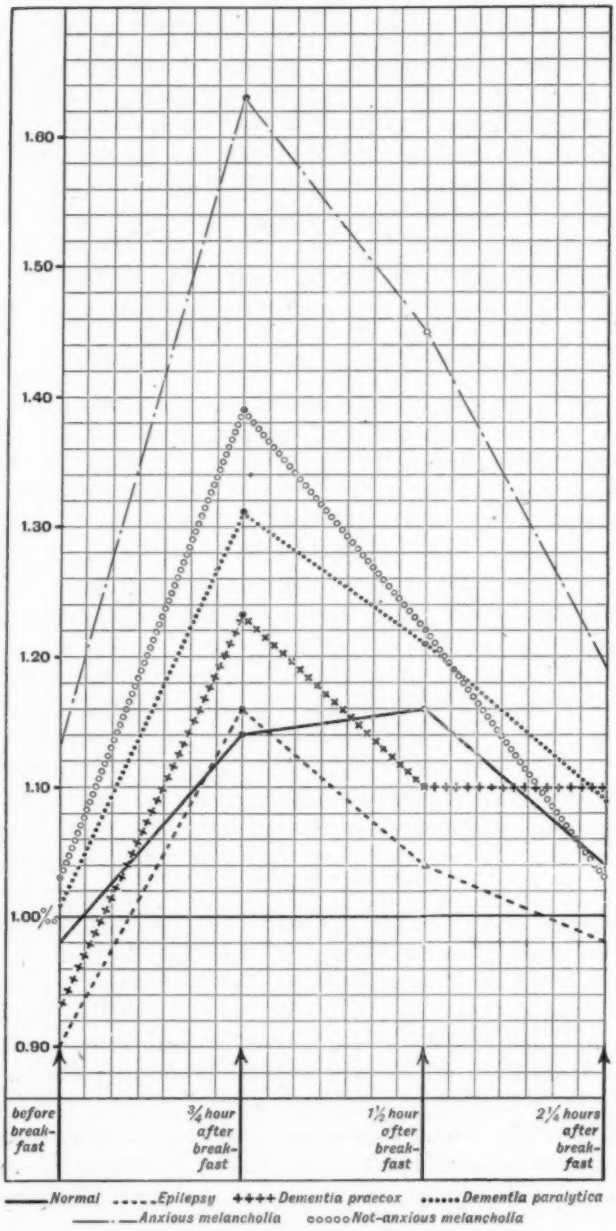
ground of this disease, which is degenerative katexogen? And is not it a remarkable fact that in this very disease we can observe so many of the old animal emotions with their appertaining bodily reactions?

Résumé.

Emotions, especially those of a depressive character, cause an increase of blood-sugar. They therefore have a deteriorating influence on diabetes mellitus. They may cause a temporary hyperglycæmia and even glycosuria in normal persons, not suffering from saccharine diabetes. We can no longer stick to any fixed limit of the amount of sugar in the blood, above which we diagnose diabetes. It is the same as with the body-temperature. It is incorrect to consider a temperature of 37° or even 37·5° C. abnormal. There are many persons —and especially those of a neuropathic constitution—who have a higher temperature (e.g., 37·8°, 37·9° C.) without any bodily reason. In the same way, even a considerable increase of blood-sugar need not be a symptom of disordered metabolism. One often gets the impression that these persons temporarily (emotions) or permanently (e.g., in the tropics) live on a higher level; in that case, glycosuria does not appear—though the blood-sugar is increased to such a degree as to cause glycosuria in normal condition—for the high amount of blood-sugar means no abnormal condition in the organism.

In the same way, hyperglycæmia may occur with any emotional state in mental disorders; this has been found by various observers and also by myself in emotions with patients suffering from general paresis, epilepsy, dementia præcox, traumatic neurosis and neurasthenia. Glycosuria and hyperglycæmia are most frequent in melancholia.

The present investigation has proved that hyperglycæmia before breakfast (spontaneous hyperglycæmia) is not a constant symptom in melancholia, but this symptom is constantly found after the patients had taken ± 200 c.c. of milk and ± 100 grm. of bread and butter (alimentary hyperglycæmia). It was shown that with marked anxiety there was the highest amount of sugar in the blood. The figure on p. 281 shows the various curves of blood-sugar before breakfast, three-quarters of an hour after breakfast, one and a half hours after breakfast, and two and a quarter hours after breakfast. The curve of epileptic dementia shows a rather low amount; that of hebephrenia only surpassed the normal curve three-quarters of an hour after breakfast. The curve of paralytic dementia is doubtless


1.60
1.50
1.40
1.30
1.20
1.10
1.00‰
0.90
before breakfast
¾ hour after breakfast
1½ hour after breakfast
2¼ hours after breakfast
Normal
Epilepsy
Dementia praecox
Dementia paralytica
Anxious melancholia
Not-anxious melancholia

higher than the normal one; probably the anatomical alterations of this disease are responsible for the increase. A high and steep curve is that of melancholia; the curve of anxious melancholia is the only one that starts much higher than the normal curve; there is a marked difference between the anxious and the not anxious forms of melancholia. No curves are given of the amount of blood-sugar in amentia and in mania; amentia is so little an entity and the percentage of sugar in the blood of maniacal patients is too variable. As for the reason of this variability, I must refer to my discussion of it in Chapter I (pp. 246 and 251); suffice it here to state that mania also shows a distinct increase of blood-sugar in highly emotional states, in anger and irritability.

It is shown by Cannon, Shohl and Wright that in animals strong emotions, such as fear and anger, cause glycosuria; this glycosuria is the direct consequence of an increase of sugar in the blood, and this hyperglycæmia again is caused by a hypersecretion of adrenalin. Cannon has pointed out that hyperglycæmia is not the only consequence of the increased adrenalin in the blood (or, which is the same thing, of the over-excitement of the sympathetic system), but that in the major emotions, in anger, fear and pain, the total sympathetic division is hyper-stimulated. All these sympathetic reactions mean so many useful things to the animal in danger.

Gaskell divides the nervous system, the involuntary as well as the voluntary one, into a somatic and a splanchnic part. The motor part of the somatic division would in this way supply the skeletal muscles and the unstriped musculature belonging to the sympathetic (thoracico-lumbar outflow). Combining the physiological conception of Cannon with the anatomical conception of Gaskell, we may come to the natural hypothesis that in moments of danger the whole somatic nervous system comes into action, whereas the splanchnic system serves for conservation of the body, or, to express it simply: the somatic division serves the outer man, the splanchnic division serves the inner man.

Cannon found many symptoms of over-excitement of the sympathetic division in emotion, all of which were useful to the animal. When we look for the other reactions on sympathetic stimulation, it appears that they are all present in emotion and they may all be of use. It is easily understood that emotions such as anger and fear have preserved their old animal reactions in man also, and that these reactions are most frequently found in melancholia. Indeed, we may find in melancholia many symptoms of over-excitement of the sympathetic

division, increase of blood-sugar, high blood-pressure, dilatation of the pupils, diminishing of secretions and inhibition of the intestinal movements.

These observations lead to a further conclusion. The deeper cause of melancholia, and especially of maniacal-depressive insanity, of periodical melancholia, of the circular type of the lighter form, the so-called cyclothymia, in short, of all those degenerative psychoses in which we find that inexplainable variability of mood, must be sought in the animal world. In this way, the frequency of the old animal reactions in melancholia may be better understood.

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